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BELL'S PALSY

Pathology and Surgery

A Report Concerning Fifty Patients Who Were Operated on After the Method of Ballance and Duel

KARSTEN KETTEL, M.D.

HILLERÖD, DENMARK

BY BELL'S palsy is understood an apparently idiopathic (genuine) peripheral disease of the facial nerve which unfortunately has been called "rheumatic facial paresis" or "paresis nervus facialis 'e frigore'."

Little is known about the disease from the standpoint of etiology; so much seems to be clear, however, that "Bell's palsy," which is far from designating an entity, has become a collective diagnosis for all cases of peripheral facial paresis in which it has been impossible to demonstrate a local causation.

The lack of knowledge has also left its mark on the discussion of the genesis of the disease, which practically has been confined to theoretic deliberations.

In 1932, however, Ballance and Duel [1] began to operate on patients suffering from Bell's palsy, decompressing the facial nerve by opening the fallopian canal from the stylomastoid foramen to the lateral semicircular canal, their view being that the disease is due to edema of the facial nerve, which is compressed by the inelastic bony canal.

By this autopsy in vivo, new ways were paved to the study of the disorder. The present publication, which is based on observations of 50 patients with Bell's palsy who were operated on in the manner of Ballance and Duel, represents an attempt to procure further knowledge, and in this attempt I have endeavored to have the following three points in view: (1) if possible, to differentiate etiologically certain groups of cases of Bell's palsy from others; (2) to arrive at a deeper understanding of the pathogenesis, and (3) to estimate the therapeutic value of the decompression operation.

All the microscopic examinations were made by Svend Petri, M.D., head of the Pathologic-Anatomic Institute of the Municipal Hospital of Copenhagen.

1. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryng. **15**:1 (Jan.) 1932.

I

PATHOLOGY

PREVIOUS INVESTIGATIONS

Microscopic Observations.—Owing to the markedly benign nature of the disease, it is only in rare instances that the opportunity is offered to examine microscopically the facial nerve of a patient suffering from Bell's palsy, and in only 4 cases have microscopic observations been described; these reports originate, all of them, from the years about 1900.

Minkowski[2] (1892) examined the facial nerve of a man aged 27, who died of hydrochloric acid poisoning. The last eight weeks before his death he had been suffering from massive Bell's palsy with the complete reaction of degeneration. The microscopic examination revealed far advanced degenerations of medullary sheaths and axis-cylinders, most marked in the peripheral parts and in the lower part of the fallopian canal, decreasing toward the geniculate ganglion; proximad to the latter the nerve was normal. It was emphasized especially that there were no signs of inflammation in or about the nerve, nor was any compression observed in the fallopian canal.

Dejerine and Théohari[3] (1897) examined the facial nerve of a woman aged 81, who died of pulmonary complications after a cancer of the uterus. The paresis was massive, and the maximal duration was four months, the minimal duration being two months. The reaction of degeneration was positive. In this case, too, a considerable parenchymatous neuritis was demonstrated, also most marked in the peripheral part of the nerve. The latter was not compressed in the fallopian canal.

Mirallié's[4] patient (1906) was a man aged 76, who died of pneumonia. During the last month he had suffered from Bell's palsy, and the microscopic examination revealed intense parenchymatous neuritis with necrosing medullary sheaths and completely degenerated axis-cylinders. As in the cases mentioned, the alterations were most marked in the more peripheral part of the facial nerve, but manifest degenerations were found also in the facial nucleus of the same side, for the greater part of the cells were voluminous, their edges had disappeared, the protoplasmic offshoots were hardly visible, and distinct chromatolysis was observed in the nuclei.

Alexander[5] (1902) made a postmortem examination of a man aged 56. The medullary sheaths had become decomposed into globules and drops, the axis-cylinders had been completely destroyed as far as the greater part was concerned, whereas the perineurium and the endosteum were natural without any signs of inflammation. In the geniculate ganglion and in the part of the facial nerve which is enclosed in the facial canal there was some round cell infiltration,

2. Minkowski: Zur pathologischen Anatomie der rheumatischen Facialislähmung, Arch. f. Psychiat. **23**:586, 1891.

3. Dejerine, J., and Théohari, A.: Un cas de paralysie faciale périphérique dite rheumatismale, ou à frigore, suivi d'autopsie, Compt. rend. Soc. de biol. **4**:1033, 1897.

4. Mirallié, M. C.: Paralysie faciale périphérique: Autopsie, Rev. neurol. **14**:702, 1906.

5. Alexander, G.: Zur Klinik und pathologischen Anatomie der sogenannten rheumatischen Facialislähmung, Arch. f. Psychiat. **35**:778, 1901-1902.

but only along the vessels of the endoneurium. There were no traces of inflammatory alterations in the bony canal proper. In the nerve of the right side the conditions were natural.

Hoffmann's [6] patients (1894) suffered from polyneuritis cerebralis and had a positive Wassermann reaction; hence they do not belong to the group of patients with true Bell's palsy.

It appears concordantly from these four, the only, microscopic examinations that in Bell's palsy investigators have found purely degenerative alterations of axis-cylinders and medullary sheaths, most marked in the peripheral parts of the nerve, whereas regular inflammatory products have not been demonstrated. Alexander mentioned only that in part of the facial nerve, and only along the vessels of the endoneurium, there was some round cell infiltration. All the authors emphasized in agreement that the alterations were most marked in the peripheral parts of the nerve, but signs of degeneration were noted in the facial nuclei also.

Minkowski and Dejerine and Théohari expressly stated that no edema could be demonstrated in the facial canal, but then it must be borne in mind that the temporal bones were not removed until several hours after the occurrence of death, and under such circumstances, undoubtedly, even severe edema may subside.

It is interesting to supplement these communications with the results of two microscopic examinations of facial nerves originating from patients suffering from otogenous pareses.

Darkschewitsch and Tichonow [7] (1893) examined a woman aged 59, who died of erysipelas and who had suffered from massive otogenous facial palsy of one year's duration. There was a complete reaction of degeneration. There were extensive areas of destruction in the tympanic cavity and in the mastoid process, but the facial canal itself was uninjured. In the microscopic examination, only slight alterations of the nerve were seen for the first 2 or 3 mm. in the peripheral direction from the geniculate ganglion; after an almost knife-edged transition of scarcely 1 mm., foci of violent degeneration of medullary sheaths and axis-cylinders were found distad from this part, so that the sheaths of Schwann were completely empty. There was no interstitial inflammation in capillaries or in the intercellular substance; in the facial nuclei *einfache Atrophie* [simple atrophy] was demonstrated.

Flatau [8] (1897) made a postmortem examination of a man aged 34, who died of pulmonary phthisis and who suffered from otitis media chronica tuberculosa sinistra, loss of hearing, and massive homolateral facial palsy with a positive reaction of degeneration. He examined both the facial nuclei and the nerve

6. Hoffmann, J.: Zur Lehre von der peripherischen Facialislähmung, Deutsche Ztschr. f. Nervenh. **5**:72, 1894.

7. Darkschewitsch, L., and Tichonow, S.: Zur Frage von den pathologisch-anatomischen Veränderungen bei peripherer Facialislähmung nicht specifischen Ursprungs, Neurol. Centralbl. **12**:329, 1893.

8. Flatau, E.: Peripherische Facialislähmung mit retrograder Neurondegeneration: Ein Beitrag zu der normalen und pathologischen Anatomie der Nn. facialis, cochlearis und trigeminus, Ztschr. f. klin. Med. **32**:280, 1897.

peripherally from the stylomastoid foramen (but not the part of the nerve enclosed in the fallopian canal) and observed marked degenerative alterations in both of these parts.

In these 2 cases of otogenous paresis exactly the same findings were made as in Bell's palsy: purely degenerative alterations of the nerve and the nuclei but no signs of inflammation, a fact that will be dealt with in detail in the following pages.

Macroscopic Observations.—Based on the view that Bell's palsy is due to a compression of the facial nerve in the fallopian canal, Ballance and Duel, in 1932, introduced the decompression operation and thus paved new ways to the study of the paresis. Slitting open the nerve sheath of a patient who had suffered from Bell's paresis for a fortnight, Duel [9] observed how the nerve bulged to twice its size, and in a paresis of two years' standing, which had subsided only partially, Duel and Tickle [10] found atrophic alterations: "The sheath was slit open. A few strands were found to be running through."

Morris [11] made the same observations: edema of the nerve in fresh paresis, atrophic alterations in inveterate paresis; thus, in an instance of eight years' standing he found the nerve transformed into a fibrous cord provided with small nodules. Horgan [12] operated on a patient with paresis of a fortnight's standing and noted that when the sheath was slit open the nerve bulged.

Whereas all of the authors mentioned to this point have been engaged in presenting the condition of the nerve, Barraud [13] and Chéridjian [14] have described some alterations in the mastoid process.

In 2 patients, a child aged 3 years and a woman aged 32, with massive facial paresis preceded by aural pain but with normal otoscopic findings, Barraud made the following observation on operation, which in both cases was performed on the fifth day of the disease: *Cellules de la pointe et de la region de nerf facial situées en dessous de l'aditus remplies de granulations grises* (cells of the apex and of the region of the facial nerve, situated underneath the aditus, filled with gray granulations). He made no comments on the facial canal proper, which was opened for 3 to 5 mm.

9. Duel, A. B.: Clinical Experiences in Surgical Treatment of Facial Palsy by Autoplastic Nerve Grafts, Arch. Otolaryng. **16**:767 (Dec.) 1932.

10. Duel, A. B., and Tickle, T. G.: The Surgical Repair of Facial Nerve Paralysis, Ann. Otol., Rhin. & Laryng. **45**:3, 1936.

11. Morris, W. M.: Surgical Treatment of Bell's Palsy, Lancet **1**:429, 1938; Surgical Treatment of Facial Paralysis, ibid. **2**:558, 1939; Surgical Treatment of Facial Paralysis, ibid. **2**:1172, 1936.

12. Horgan, J. B.: Bell's Palsy Treated by Incision of Sheath of Facial Nerve, Brit. M. J. **2**:768, 1839.

13. Barraud, A.: Paralysies du nerf facial "å frigore," Ann. d. mal. de l'oreille, du larynx **45**:564, 1926.

14. Chéridjian, Z.: À propos de deux cas de paralysie faciale dite "a frigore," Schweiz. med. Wchnschr. **74**:386, 1944.

On the other hand, in a boy aged 13, who underwent operation on the twentieth day of the paresis, the bony system was found to be completely normal.

Chéridjian has also operated on 2 patients suffering from Bell's palsy. In the first patient the paresis was accompanied by slight hyperemia of the tympanic membrane and moderate exudation in the middle ear. The hearing was decreased, with marked buzzing in the ear; there was slight tenderness over the mastoid process on pressure, and slight blurring of the cells of the mastoid process was seen on roentgen examination. After paracentesis there was a serous discharge for a couple of days. As the patient's condition, despite conservative treatment, remained unchanged fifteen days after the paracentesis, resection of the mastoid process was performed, but no decompression of the facial nerve; nothing abnormal, however, was observed in the operation. Mobility did not appear until several months afterward; the paresis did not subside completely, however, and a slight contracture developed.

The next patient was a woman aged 46, who suffered from massive Bell's palsy, pain in her ear, a highly troublesome buzzing in the ear and hemicrania; there was slight redness of the posterior wall of the auditory meatus, upward and posteriorly at the annulus tympanicus; the tympanic membrane and the hearing were normal; there were slight tenderness and swelling of the mastoid process and swelling of the retromastoid and cervical glands.

Two and a half months later the headache was still intense, and the buzzing in her ear was unbearable, but the rest of the examination did not show any changes. Faradic and galvanic reactions were positive; the reaction of degeneration was not present. Roentgen study of the mastoid process showed nothing abnormal. Resection of the mastoid process without decompression of the facial nerve was performed. No pus, no granulations were encountered, the antrum was larger than the normal and surrounded by a zone with profuse hemorrhage, *un état local très fortement congestif* (a local state of very intense congestion), near the upper bend of the facial nerve. The facial canal was not opened. Immediately after the operation the headache and the buzzing in the ear subsided completely, and the paresis at once began to abate, at last subsiding completely.

Flodgren [15] has treated a patient with peripheral facial palsy of three days' standing. Apart from a slight injection in the attic and around the processus brevis, the tympanic membrane was dull, retracted and without perforations. The hearing of both ears was reduced to comprehension of the speaking voice at 1.5 meters, the loss being of mixed type. Roentgen study showed that the region of the facial canal was more dense than that of the nonparalyzed side. A minimal drop of clear mucus escaped on paracentesis. Resection of the mastoid process was performed and, while the superficial cells and the part round the antrum were natural, three large cells, communicating with each other and containing a reddish brown viscous semisolid mass—obviously stale blood—were found in the deeper part close to the facial canal. As this blood was being removed, the patient felt the paresis improve. The author considers it probable that there was dehiscence in the facial canal. Flodgren mentions that if the patient had not come under the treatment of a specialist at once, this case would undoubtedly have been regarded as one of Bell's palsy. As a matter of fact, I think it is a case of Bell's palsy, as will be explained later. Here I only want to stress that the drop of fluid which escaped on paracentesis might be an exudate and that in the mastoid cells Flodgren [16] found stale blood but no pus or granulation tissue.

15. Flodgren, E.: Nord. med. (Hygiea) **31**:2159, 1946.

Cawthorne [16] stated that in 12 of 14 cases the nerve trunk appeared to be unduly constricted at the level of the stylomastoid foramen, the constriction being accentuated by swelling of the nerve immediately above it. The billowing out of the nerve above the constriction after incision of the sheath is, according to this author, peculiar to this condition. He used a dissecting microscope and noted at the side of the constriction several tiny hemorrhagic streaks running longitudinally. In 2 cases of more than six months' duration he found the nerve trunk in the neighborhood of the stylomastoid foramen to be shrunken, reddened and adherent to the skin.

Bergström [17] on the other hand, made a decompression in a woman who for fifteen years had suffered from Bell's palsy, which only partially had disappeared and which three months before the operation had recurred without any subsequent improvement. It is extremely interesting that after the decompression the patient regained almost full mimicry. However, with regard to the author's statement that he, in doing the decompression, found that the facial canal and the nerve from just below the lateral semicircular canal until just before the entrance to the pyramid was constricted, its width being little more than half in proportion to the part below the bend at the lateral semi-circular canal, I want to point out that I in most of my decompressions on patients suffering from Bell's palsy and also from experience in the postmortem room on cadavers that had never had a facial palsy have made the same observation. This seems to indicate that Bergström's observation is a normal anatomic finding.

Summarizing, it can be said that a few authors (Barraud; Chéridjian; Flodgren) have found distinct alterations in the cells of the mastoid process which are situated along the facial canal, whereas others (Duel; Duel and Tickle; Morris; Collier [18]; Horgan; Cawthorne) have demonstrated edema of the nerve in cases of fresh paresis, atrophic alterations in cases of inveterate paresis.

EXAMINATIONS ON WHICH THE PRESENT REPORT IS BASED

My examinations were made in the course of fifty decompression operations carried out according to the following three indications, the justification of which will be thoroughly discussed in part II of the present work:

1. Paresis which, after a period of observation of up to two to three months, had not shown any signs of incipient spontaneous mobility; this type was encountered in a total of 25 patients. In the following pages this group is termed: "Patients with fresh paresis."
2. Paresis in which the spontaneous mobility had come to a standstill before complete restitution had been attained; patients with

16. Cawthorne, I.: Peripheral Facial Paralysis: Some Aspects of Its Pathology, Laryngoscope **56**:653, 1946.

17. Bergström, E.: The Question of the Etiology and Treatment of What Is Known as Rheumatic Facial Paralysis, Acta oto-laryng. **34**:541, 1946.

18. Collier, J.: Facial Paralysis and Its Operative Treatment, Lancet **2**:91, 1940.

this type were operated on from nine months to three years after the spontaneous improvement had ceased. The group comprises 17 patients and is termed: "Patients who recovered from paresis with defect."

3. Relapsing paresis; this was observed in a total of 8 patients.

Pathologic-anatomic changes were found partly in the bony system, both in the cells and in the part of the facial canal passing from the stylomastoid foramen to the lateral semicircular canal, and partly in the nerve itself; in both groups the changes were macroscopic and microscopic.

PATHOLOGIC ANATOMY

Changes have been demonstrated both in the cells of the mastoid process and in the facial canal itself—in both parts at the same time in some cases; in others, as isolated phenomena.

Cells of the Mastoid Process.—In 39 cases there is detailed information about the condition of the mastoid cells. In the remaining 11 cases nothing is stated about the mastoid cells, which means that conditions proved to be macroscopically normal. The results of the examinations are, then, as follows: In 39 cases (28 + 11) the cells were macroscopically normal. In 5 cases there was necrosis of the cells, spreading to the entire system. In 6 cases a large focus of necrotic bone was demonstrated at the apex of the mastoid process and especially around the stylomastoid foramen, but no pus or granulations.

These changes were distributed within the three groups of patients with facial palsy as follows:

Patients with fresh paresis (massive in all of them): in 21, macroscopically normal cells; in 4, destroyed cells.

Patients recovered from paresis but with defect: in 14, macroscopically normal cells; in 3, destroyed cells.

Patients showing relapse of paresis: in 4, macroscopically normal cells; in 4, destroyed cells.

The case records of the 11 patients with changes of the mastoid cells are reported in the following pages.

CASE 1.—A woman aged 46 was admitted on Nov. 6, 1945. Thirteen years ago she had acute suppurative otitis media on the right side. The ear had been dry since that time; the hearing, good. Two and one-half months before admission a massive right-sided peripheral facial palsy developed in the course of twenty-four hours, preceded by ten days' pain in her forehead and temple, radiating down behind her right ear; it was accompanied by gustatory disturbances and one day's dizziness but not by pain in the ear itself or by discharge of the latter; there were no disturbances of hearing and no buzzing in the ear, no eruption of herpes zoster and no increase of temperature. The paresis was treated with Insipidin (powdered posterior pituitary) and injections of vitamin B without effect. The paresis was still massive; the faradic reaction was negative and the galvanic reaction positive. Downward posteriorly on the right tympanic membrane there

was a large atrophic part. There was just a suspicion of changed consistency and tenderness of the right mastoid process. Otoscopy and acousticovestibular examination indicated natural conditions. Roentgenologically, there was slight blurring of the cells on the right side, and behind and lateral to the antrum the limits of the cells were indistinct. The sense of taste was suspended for all the four qualities on the foremost two thirds of the tongue. The remainder of the examination, including the neurologic phase, showed normal conditions. The Wassermann test was negative. On November 8, decompression of the right facial nerve was performed, with the following note being recorded: "The cellular system is highly irregular, the cells dark colored and beyond all doubt soft around the antrum and between the posterior wall of the auditory meatus and the sinus. As the nerve sheath is slit open, the nerve bulges enormously, protruding like a hernia."

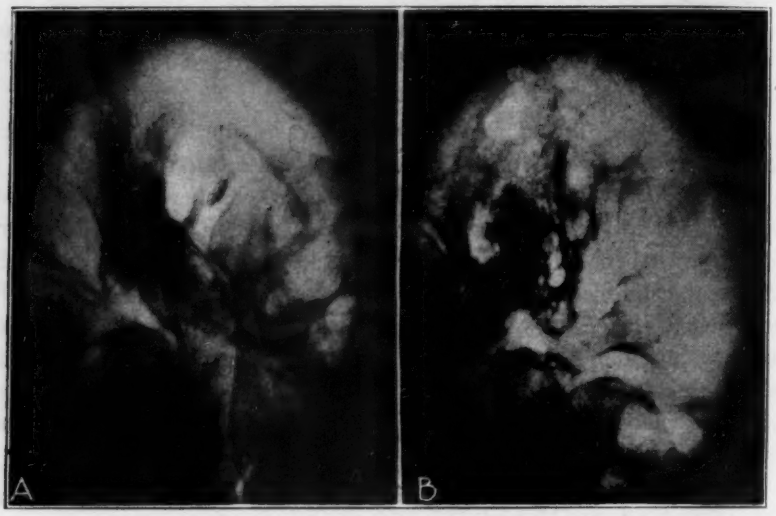

Fig. 1 (case 2).—Roentgenograms of a patient with Bell's palsy on the right side of five months' duration. *A*, right side. The mastoidal cells on the paralyzed side are completely clouded. *B*, left side.

Microscopic examination of the mastoid cells revealed spongy osseous particles with slight fibrous transformation of the marrow and scanty, limited granular bony necrosis. There was no round cell infiltration.

Microscopic examination of the wall of the facial canal revealed more marked granular necrosis, no inflammatory infiltrates and no granulation tissue.

Case 2.—A man aged 39 years entered Rigshospitalet, in Oslo, Norway, in August 1945. He had never had suppurative otitis media. Twenty years before admission he noted left-sided facial palsy, which subsided spontaneously. Five months before admission massive right-sided facial palsy suddenly appeared, accompanied by pain in the right ear but not with discharge of the ear; there was no decrease of hearing, no dizziness. Spontaneous improvement did not occur. There was a complete reaction of degeneration. Roentgen examination revealed diffuse blurring of the cellular system of the right side (fig. 1). Otoscopic findings and hearing were normal; the remainder of the objective examination showed nothing abnormal. The Wassermann test was negative.

On August 22, decompression of the facial nerve was performed, with the following note being recorded: "Around the antrum, both laterad to and behind it, the tissue is strikingly soft and filled with a dark fluid, but no pus is found. The facial canal is also soft and is easily opened. No bulging of the nerve can be ascertained when the nerve sheath is slit open."

Case 3.—A woman aged 49 was hospitalized on Jan. 15, 1945. She had never had suppurative otitis media. Two years before admission massive left-sided facial palsy suddenly appeared, not accompanied by pain in or behind the ear or by acousticovestibular symptoms. Eight days after the onset of the paresis the tympanic membrane and the hearing were normal. The paresis subsided spontaneously in the course of a month. Four days before admission, again massive left-sided paresis appeared, accompanied by slight pain behind but not in the ear of the same side; there were no acousticovestibular symptoms, no unquestionable gustatory disturbances, no herpetic eruption and no increase of temperature. There was a very slight injection along the posterior edge of the shaft of the left malleus (but this congestion could also be observed one year after). Acousticovestibular examination showed natural conditions. Roentgen examination of the cells showed all of them to be quite small and blurred, especially toward the apex of the mastoid process. The borders of the cells could be defined only with difficulty. The Wassermann test was negative.

On Jan. 16, 1945, decompression of the left facial nerve was performed, following the diagnosis of relapsing Bell's palsy. "The osseous tissue around the antrum and in the cells between the posterior wall of the auditory meatus and the sinus is soft. The facial canal is hard. When the nerve sheath is slit open, the nerve fibers protrude forcibly."

Microscopic examination of osseous tissue taken from the antrum revealed bony fragments of normal structure with limited, superficial, slight granular necrosis and slight to moderate "cicatricial" alterations of the endosteum.

Case 4.—A woman aged 20 was admitted to a hospital on Jan. 15, 1945. She had never had suppurative otitis media. Two and a half months before admission massive left-sided peripheral facial palsy suddenly developed, with gustatory disturbances and a pricking sensation in the left ear but no discharge of the ear and no acousticovestibular symptoms. On admission the patient presented massive peripheral facial palsy. The faradic reaction was negative; the galvanic reaction, positive. Otoscopic and acousticovestibular examinations showed normal conditions. Roentgen study revealed blurring of the cells of the left mastoid process with distinct destruction at about the middle. The remainder of the objective examinations, especially the neurologic, disclosed nothing abnormal.

On January 17, decompression of the left facial nerve was performed, with the following observations being recorded: "Immediately after the cellular system is opened, it is seen to be soft, with marked 'osteitis' of the antrum, the angular cells and the cells between the sinus and the posterior wall of the auditory meatus. The mucous membrane is dark colored; no pus is seen. Around the stylomastoid foramen there are 'osteitic' alterations. Such alterations are found also in the wall of the entire facial canal; especially there is a cord of retrofacial cells which are completely soft. Before the nerve sheath is slit open, the nerve is seen to be highly edematous distad to the stylomastoid foramen, and as the sheath is slit open, the nerve protrudes as a whitish yellow, enormously edematous balloon, the continuity of which is interrupted. The nerve ends are easily brought into contact."

Mobility returned in ten months.

One cannot exclude the possibility that the interruption of the continuity was caused as the nerve sheath was slit open, despite the fact that this was done in exactly the same manner as in more than 150 similar cases, with Graefe's knife, the point of which was just introduced into the nerve sheath, with the back of the knife toward the nerve. That the continuity of the nerve may be interrupted in Bell's palsy, trauma having been safely excluded, will appear from case 5.

CASE 5.—A woman aged 30 years was admitted to a hospital on Jan. 26, 1945. She had never had otitis media. Fourteen years before admission she suddenly experienced massive right-sided peripheral facial palsy, with gustatory disturbances but without acousticovestibular symptoms. Simultaneously she had marked edema of the upper and lower lips, especially on the right side. This edema remained unchanged thereafter, whereas the paresis subsided gradually, though not completely, and slight contracture developed. Three months before admission the patient felt pain in and behind her left ear, but there was no discharge of the ear; then suddenly massive left-sided facial palsy occurred, with buzzing in the ear but without vestibular symptoms or herpetic eruption. The temperature was not increased. Since then the paresis has remained massive. The patient displayed typical Melkersson's syndrome. On her right side she had partial facial palsy, slight contracture and ticlike convulsions; on her left side, peripheral massive paresis. Both tympanic membranes were dull; the hearing was normal; the faradic reaction was positive on the right and negative on the left side, with a complete reaction of degeneration on the left side. Vestibular and ophthalmoscopic examinations showed normal conditions. The Wassermann test was negative. Examination of the spinal fluid revealed: 4 cells per 3 cu. mm. (4/3) (Fuchs-Rosenthal chamber used); albumin < 10 mg., and globulin < 1 mg., per hundred cubic centimeters. The tongue and lips were affected as in Melkersson's syndrome. The neurologic state was otherwise normal. Roentgenologically, the mastoid process showed slight blurring of the cells of the left side.

On January 29, decompression of the left facial nerve was performed, with the following observations being recorded: "The superficial cells are large and glossy. In the depth around the antrum and in the cells between the posterior wall of the auditory meatus and the sinus the bone is soft, increasingly so to the part around the stylomastoid foramen. The entire facial canal is completely necrosed in its distal half, and the remaining part is opened. Neither knife nor scissors was used; still the nerve sheath is seen to be open, and from the latter a highly edematous piece of nerve bulges out, being conically pointed downward and of a grayish pink color; the point of the nerve, however, is of a much darker red color than the remaining part. The piece of the nerve projecting from the opening in the sheath is 7 mm. long. It is resected and examined microscopically. A nerve transplant taken from the ilio-inginual nerve is grafted" (and the mobility reappeared ten months later).

The results of the microscopic examination of the osseous tissue may be outlined as follows:

1. Superficial cells: scanty, limited subperiosteal granular bony decay; bone otherwise normal, with no signs of necrosis and no round cell infiltration.
2. Periantral cells: granular decayed osseous tissue; slight necrosis; here and there small tufts of "cicatricially" changed connective tissue enclosing small groups of granular decayed bone; no inflammatory infiltration.
3. Tissue from the parts near the stylomastoid foramen: same as under 2.

The microscopic examination of the nerve and the nerve sheath was recorded as follows: "The nerve sheath consists of dense collagenic connective tissue, very

poor in cells but enclosing small vessels and nerve branches. In the connective tissue are slight 'cicatricial' alterations and a few small groups of chalky deposits. The lumen of a small venous branch contains bulging, partially connected, slightly chalky connective tissue masses (possibly a thrombus in full organization). Otherwise the connective tissue is the seat of edema and hemorrhage and is covered on the inside with remnants of a fibrinous exudate. No remnants of blood pigment and no fresh-formed thrombus is seen in the vessels. On the whole the nerve is of a uniform, moderately cellular structure; still it is somewhat loose everywhere. The nerve filaments stain slightly; they are of unequal size and not always of a regularly circular shape in transverse sections. The nerve filaments are split by slight edema, and the blood capillaries are highly dilated with blood. Here and there small fresh punctiform hemorrhages are seen, as well as small accumulations of lymphocytes, and a few small cicatricial strands. There is no emigration of leukocytes. Special staining in the manner of Mahon showed degenerations of the medullary sheaths; moreover, there is staining of axis-cylinders, which appear to be distinctly degenerated."

On February 2, decompression of the right facial nerve was performed. The cellular system, the facial canal and the nerve were macroscopically normal. Microscopically, the osseous tissue also showed normal conditions.

Thus in 5 cases macroscopic necrosis of the entire cellular system was seen, especially marked in the apex of the mastoid process. In all these cases the necrosis could be verified by roentgenogram. The bone was completely soft, and in 1 case it contained a dark clear liquid.[19] The bony tissue could be removed as a soft mass by means of a sharp spoon exactly as in well defined mastoiditis, but no pus and no granulations were found. After the excochleation the distal part of the mastoid process had been transformed into a deep cavity.

The microscopic examination showed slight halisteresis of the bony substance, marked granular decay of the bone and fully developed bony necrosis, while the covering membranes ("endosteum") were thickened, containing few cells and vessels and being hyalinized, whereas remains of granulation tissue or chronic round cell infiltrates could not be demonstrated. The outstanding features are thus necrosis and exudation but no round cell infiltration and no granulation tissue.

In the following 6 cases the pathologic alterations could be found only around the stylomastoid foramen, whereas the remaining cellular system was normal, especially around the antrum.

CASE 6.—A woman aged 37 was admitted on June 26, 1944. Three years before admission she was suddenly seized with massive right-sided peripheral facial palsy. This had been preceded by pain behind but not in the right ear. There had been no acousticovestibular symptoms, no eruption of herpes zoster, no increase of temperature. The paresis gradually subsided in the course of the next two years, but thereafter the condition was stationary. Mobility was fairly good in the region of the second branch, but was only slight in the region of the third branch and was completely suspended in the region of the first branch. Otoscopic, acoustico-

19. In another patient, recently operated on, the entire cellular system was necrotic and filled with a clear dark fluid.

vestibular and roentgen studies of the ears and the remainder of the objective examinations, including the neurologic, showed normal conditions.

On June 27, decompression of the right facial nerve was performed, and the following observations were recorded: "The cellular system is large and extensive, but all the superficial cells are completely glossy. In the vicinity of the stylomastoid foramen the bone is soft, and a plunge is made into a cavity, which is a little over the size of a hazelnut and lies in the part deep to the facial nerve, toward the bulb of the jugular vein. In the anterior wall of this cavity the facial nerve is completely exposed; it is spindle shaped and has been transformed into a cord resembling connective tissue."

Microscopic examination of the osseous tissue showed spongy osseous tissue of normal structure with normal red marrow and normal periosteum.

Case 7.—A woman aged 24 entered Rigshospitalet, in Olso, Norway, in August 1945. She had never had suppurative otitis media. Three years before admission massive right-sided peripheral facial palsy suddenly developed, preceded by pain behind but not in the ear. There was no discharge of the ear, and no acousticovestibular symptoms. Her condition improved a good deal in the course of the first six months but thereafter was stationary. Two years before admission massive left-sided peripheral paresis developed with exactly the same facies morbi as on the right side. In this, too, improvement continued for the first six months and no improvement occurred thereafter. There was then bilateral paresis, most marked on the right side, with greatly decreased, though not completely suspended, function of all the three branches. Roentgen study of the ears, otoscopy and acoustic examination showed normal conditions. There was distinct quantitative reduction but no qualitative alteration of electric irritability. The remainder of the objective examinations showed normal conditions.

On August 27, decompression of both facial nerves was performed. All the superficial cells on both sides looked natural. But around the stylomastoid foramen the bone was completely soft and of an almost dark red color. When the nerve sheath was slit open, the nerve bulged considerably. No record of a microscopic examination is available.

Case 8.—A woman aged 57 years was admitted on May 1, 1944. She had never had suppurative otitis media. Nine months before admission massive peripheral right-sided facial palsy developed suddenly. It was not accompanied by acousticovestibular symptoms. The paresis subsided partially, but contracture and marked facial spasm developed on the paretic side. On admission there was marked contracture on the right side, with decreased strength in the whole of the part innervated by the facial nerve; at short intervals there were intense hemispasms in the parts innervated by the second and third branches. Otoscopy, acousticovestibular examination, roentgen study of the ears, and the remainder of the objective examinations, including the neurologic, showed nothing abnormal. The Wassermann test was negative.

On May 2, decompression of the right facial nerve was performed, and the following observations were recorded: "The superficial cells are large and clear, whereas the cells between the posterior wall of the auditory meatus and the sinus are smaller and soft, and it appears that the facial canal proper is so soft that the decompression is completed in a few minutes. In the part deep to the nerve a cavity, larger than a hazelnut, is found after the excochleation, and through this cavity the nerve has been running practically free. The cavity passes deeply toward the jugular foramen. The nerve sheath is slit open, and the nerve bulges distinctly."

Microscopic examination revealed minimal granular osseous tissue, surrounded by "cicatricial" connective tissue.

CASE 9.—A man aged 26 was admitted on Dec. 8, 1944. He had never had suppurative otitis media. Sixteen years before admission he suddenly experienced massive right-sided peripheral facial palsy, which subsided completely in the course of a few months. Six years later a relapse occurred, which subsided only partially. One year prior to admission massive left-sided, peripheral facial palsy suddenly appeared, not accompanied by acousticovestibular symptoms or pain. To begin with, there was improvement, but for two months prior to admission his condition had been stationary. Facial function was decreased on both sides, most strikingly on the left side. Otoscopy, acoustic examination, roentgen study of the ears and the remaining objective examinations showed normal conditions.

On December 11, decompression of the left facial nerve was performed, and the following record was made: "A large cellular system is disclosed, which is macroscopically normal. The external shell of the facial canal is enormously hard, but the interior is soft. The nerve is distinctly swollen and bulges greatly after the nerve sheath has been slit open."

Microscopic examination revealed slight granular decay of the wall of the facial canal and doubtful incipient bony necrosis.

On December 27, decompression of the right facial nerve was performed, with the following observations being recorded: "The superficial cells are completely normal. The nerve is sought out at the stylomastoid foramen, and more than half the way to the lateral semicircular canal a focus is reached in the part deep to the facial canal. Here the bone is extremely soft and is scraped out like butter. After removal of all decayed osseous tissue, the bulb of the jugular vein has practically been reached. The cavity passes 1.3 cm. below the nerve, measuring 2.5 cm. proximodistally, and off the course of the nerve in the facial canal the funnel-shaped cavity has a width of 0.5 cm. When the nerve sheath is slit open, the nerve bulges distinctly."

Microscopic examination revealed enchondromatous alteration of spongy osseous tissue, doubtful, incipient scanty bony necrosis and slight granular decay.

CASE 10.—A woman aged 29 was hospitalized on Nov. 23, 1943. She had never had suppurative otitis media. In the course of the last nine years before admission she had noted facial palsy six times, the left and the right side being involved alternately, and she believed that the attacks set in when she had been exposed to cold. They always began with intense boring pain behind the ear, and after the course of two to three days the palsy was manifest, reaching its maximum in the course of twenty-four hours. There were no acousticovestibular symptoms, no gustatory disturbances, never any rise of temperature and no herpetic eruption. The first times the paresis subsided completely, but later a partial defect remained on either side, which influenced her psychically to an extremely high degree. She had a masklike face and bilateral facial palsy. The forehead could be wrinkled a little on the right side, and the eyes could be closed; there was only slight mobility of the mouth, mostly on the left side. Otoscopy, tests of acoustic function and the remainder of the general examination showed normal conditions. Roentgen examination showed the cells of the right side to be smaller than those of the left side, but all the septums were sharply outlined. Apparently there was blurring of the cells around the antrum and down along the posterior wall of the auditory meatus, but it was somewhat difficult to judge, as the cells here were so much smaller than those of the left side.

On November 26, decompression of both facial nerves was performed, and the following observations were recorded: "On the right side the cellular system is

found to be rather changed. The facial canal seems to be soft, and after the decompression has been completed, the nerve is suspended in the whole of the posterior wall of the auditory meatus as an entirely free thread. The nerve sheath is slit open, and the nerve is now seen shining white, but it does not bulge. On the left side the cellular system is larger, and the cells are completely clear [nothing is stated about the facial canal]. The nerve is sought out at the stylomastoid foramen, and now that both sides can be compared, it is evident that the right nerve is atrophic. The nerve of the left side is far more edematous and swollen, and bulges very much as the nerve sheath is slit open."

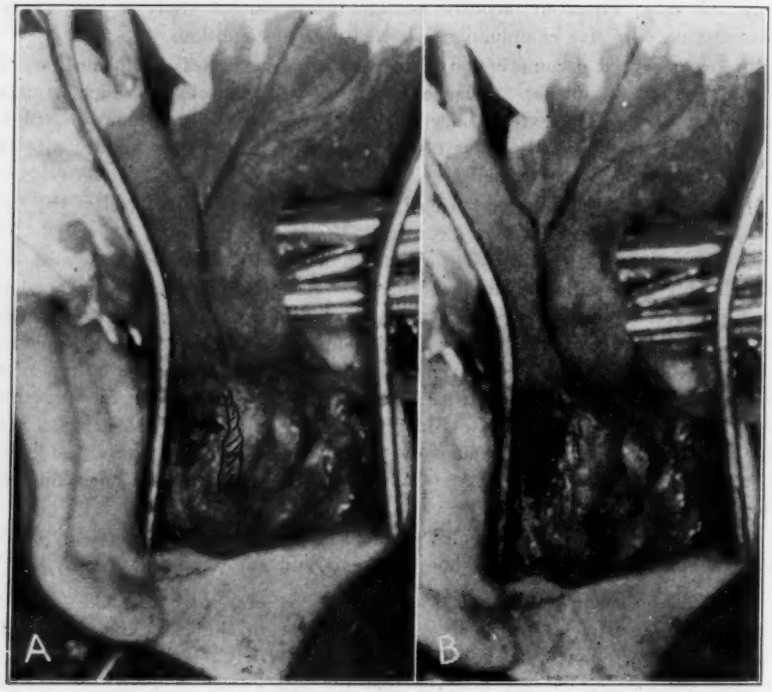

Fig. 2 (case 10).—The facial nerve is decompressed from the stylomastoid foramen to the lateral semicircular canal. In *A* the nerve is hatched. The nerve sheath is split up to the arrow. It may be seen how the nerve bulges after splitting. The same is shown in *B*, but without hatching of the nerve.

In figure 2 the left nerve is seen to be exposed from the stylomastoid foramen to the lateral semicircular canal, and the nerve is split over the distal half, not over the proximal half.

CASE 11.—A woman aged 30 entered Øresundshospitalet, Elsinore, Denmark, on Jan. 9, 1945. At the age of 3 years she had had massive peripheral facial palsy, not accompanied by any symptoms from the ear. It subsided completely, but at the age of 17 she again had left-sided peripheral facial palsy, preceded by pain in the ear for a couple of days. Paracentesis was performed, and there was slight secretion for a couple of days. Thereafter she had facial palsy on the left side six times, accompanied by pain behind the ear but not by acousticovestibular symptoms apart from slight buzzing in her ear. There was no discharge of the ear, no

herpetic eruption, no rise of temperature. Each time the partial defect as regards mobility increased, and the patient was severely influenced psychically by all her attacks of paresis. The function of the nerve was greatly reduced as far as all the three branches were concerned. Otoscopy showed the tympanic membrane to be dull and gray, but without defects.

The remaining objective examinations showed normal conditions. No roentgen study was made.

On January 10, decompression of the left facial nerve was performed, and the following note recorded: "The superficial cells are completely natural, glossy and clear, but around the stylomastoid foramen the bone is soft, and the decompression takes place extremely rapidly. The nerve is somewhat swollen, and it can be seen to press like a hernia before its entire circumference has been exposed. When the nerve sheath is slit open, the white nerve fibers protrude forcibly. At last an extensive cavity is found in the part deep to the facial nerve."

Microscopic examination revealed moderate granular decay of the bone and small hyalinized parts of connective tissue displaying slight "cicatricial" alteration and partly surrounding the granular decaying bone.

It is characteristic of the last 6 cases (6 to 11) that a cellular system was found whose greater part was macroscopically normal and that at the apex of the mastoid process and particularly around the stylomastoid foramen so large a decay of the bone was found that when it had been curetted the entire distal part of the mastoid process had been transformed into a large cavity passing deeply into the parts below the facial nerve. In case 6 the nerve was lying exposed in the anterior wall of such a focus.

In the microscopic examination granular decay of the bone, particularly bony necrosis, was demonstrated, and in a single case, enchondromatous alteration of spongy osseous tissue, but no pus and no round cell infiltration.

In this condition it may be of interest to call to mind a case which clinically resembled one of relapsing Bell's paresis completely and in which otoscopy, tests of acoustic function and roentgen study of the ears showed normal conditions. In the decompression a quite similar cavity was found around the stylomastoid foramen, filled with "granulations," which in the microscopic examination proved to be neurinoma (Kettel,[20] 1946).

The question is whether these changes can be diagnosed roentgenologically. Racoveanu [21] has contributed a case of Bell's palsy in which blurring of the mastoid cells could be ascertained by means of a roentgenogram. In my first 5 cases, in which the decay had spread to the

20. Kettel, K.: Facial Palsy of Otitic Origin, Arch. Otolaryng. **37**:303 (March) 1943; Surgical Therapy of Otogenic Paralysis According to Ballance-Duel and Bunnell, Nord. med. (Hospitalstid.) **24**:1783, 1944; Neurinoma of the Facial Nerve, Arch. Otolaryng. **44**:253 (Sept.) 1946.

21. Racoveanu, V.: Mastoïdite primitive latente débutant par une paralysie faciale, Rev. de laryng. **53**:1186, 1932.

entire cellular system, slight to more intense blurring of the whole of the cellular system could be observed, and in a few of the plates destruction of cellular septums could also be seen. In 6 cases changes were found only around the stylomastoid foramen. In 5 of these roentgenograms were made, but only in one of them could slight blurring be demonstrated.

The Facial Canal.—In 18 cases the facial canal was completely soft, so that the opening of the latter, normally a hard and time-consuming work, was completed in a few minutes. In a few cases, the outer shell of the canal was hard, and the internal layer, on the other hand, was quite soft.

These changes were distributed within the three groups of patients with facial palsy as follows:

Patients with fresh paresis: in 18, canal natural; in 7, canal soft.

Patients recovered from paresis but with defect: in 10, canal natural; in 7 canal soft.

Patients showing relapse of paresis: in 4 canal natural; in 4, canal soft.

Microscopic examination of the soft osseous tissue removed from the facial canal revealed: (*a*) slight to more marked decay of the osseous tissue proper; (*b*) "cicatricial" thickening of the covering membranes, which in part adhered to the bone, in part enclosed remnants of granular decayed osseous tissue.

In some cases these microscopic changes were distinct; in other cases they were but slightly visible.

Lastly, if the relation between the changes of the mastoid cells and those of the facial canal is examined, it appears that in 7 of the 11 cases in which there was destruction of the cells there was also displayed decay of the facial canal, but in no less than 15 cases decay could be demonstrated in the facial canal proper while the cellular system appeared natural.

With a view to control of the foregoing observations, the mastoid cells in 20 per cent of the cases were compared with macroscopically normal mastoid cells originating partly from postmortem material selected at random (25 cases) and partly from patients operated on for otosclerosis (10 cases). It appears from these comparisons that the osseous changes observed by me in Bell's palsy are pathologic, as they could not be observed in the control material, but the changes of the covering membranes (thickening of the endosteum, "cicatricial" changes) were also noted in the control material, in which they were as frequent and as pronounced as in cases of Bell's palsy. This means that the endosteum normally is rather thick in this region, or else that a slight infection has taken place at some time or other without giving any clinical symptoms, just as cicatricial changes may be noted in the tonsils or the appendix in persons who have never displayed any clinical

symptoms. As they, however, were found in the control material too, they are of no significance whatever for the pathology of Bell's palsy, being a normal or a pathologic finding. Still it was astonishing to see how slight the necrosis might be microscopically, even in the cases in which there was macroscopically extensive decay.

In order better to understand this discrepancy, microscopic examinations were made of osseous tissue from 5 patients with chronic and 15 patients with acute mastoiditis, this having occurred in association with suppurative otitis media in all of these patients. The examinations showed that even in the severest cases of acute mastoiditis of two to three weeks' standing, in which the cellular system had been transformed into one porridge-like mass of bone, the osseous trabeculae had maintained their structure amazingly well as observed with the microscope. Thus there was no proportionality between the operative findings and the microscopic picture. Hence, the comparatively well preserved bony cells from the 11 patients suffering with Bell's palsy cannot surprise one, despite the macroscopically considerable decay.

Thus it must be concluded that in 20 per cent of the cases of Bell's palsy bony necrosis of the mastoid cells, especially around the stylomastoid foramen, has been demonstrated, but no signs of round cell infiltration or granulation tissue. In 36 per cent of the cases similar changes were, moreover, found in the wall of the facial canal.

The Facial Nerve.—As already mentioned, Ballance and Duel, Morris and Collier have pointed out that the part of the facial nerve passing through the fallopian canal is edematous in Bell's palsy, but nobody has tried to substantiate the frequency of this by means of statistics or case records. In the nature of things, microscopic descriptions of the nerve itself are available in few cases only, and all are based on postmortem preparations, but they show without ambiguity that one has to do merely with phenomena of degeneration; the presence of regular inflammatory products could not be demonstrated.

When the findings are being considered, the lack of a suitable control material is again conspicuous. Even normally the nerve is said to bulge a little when the sheath is slit open—but just how much? It is true that on decompression of the facial nerve of a patient suffering from Bell's palsy I have observed, on several occasions, that the nerve did not bulge when the sheath was slit open, but this might indicate incipient atrophic alterations, and it is of no use as a control. More suitable for this purpose are 3 cases in which otogenous paresis came under my treatment immediately after a radical operation in which the nerve had been injured in its course across the promontory. In these cases I opened the entire facial canal, but when the nerve sheath was slit open, the nerve did not bulge. Larsen [22] in 3 cases made the same observation.

The alterations in a great number of patients were, however, so considerable that after the sheath had been slit open the nerve increased to two to three times its former thickness, but one cannot avoid the observation that there is a borderland in which the estimation is uncertain.

Within the different groups of patients with facial palsy the alterations were distributed in the following manner:

Patients with fresh paresis (massive in all of them; average duration two to three months): in 1, nerve possibly atrophic; in 5, nerve macroscopically normal; in 4, nerve slightly edematous; in 15, nerve distinctly edematous.

Patients recovered from paresis but with defect (paresis previously massive; duration nine months to three years): in 1, nerve apparently atrophic; in 4, nerve macroscopically normal; in 5, nerve slightly edematous.

Patients showing relapse of paresis: in 2, nerve macroscopically normal; in 6, nerve distinctly edematous.

Examples of such conditions have already been given in the foregoing case reports.

If all doubtful and weak reactions are rejected, there are still, out of 50 cases, 28 in which there was no doubt at all that macroscopically the nerve was highly edematous, and it is interesting to note that the edema could be demonstrated particularly in the fresh and in the relapsing paresis.

In 1 case the changes in the bony system and in the nerve itself were so violent that the continuity of the nerve was interrupted, and for this reason a piece of it was resected for microscopic examination, as has been described (case 5, page 436).

EPICRISIS

Briefly summarized, the results of the pathologic examinations made by me in cases of Bell's palsy are as follows:

The Bony System.—An extensive bony necrosis was demonstrated macroscopically in the mastoid cells of 11 patients, or 20 per cent. In 5 cases it had spread to the entire cellular system and could be demonstrated by means of roentgenograms, whereas in 6 cases it was found only in the apex of the mastoid process and especially around the stylomastoid foramen. Only in 1 of the latter cases could the focus be demonstrated by means of a roentgenogram. In some of the cases a clear dark fluid was observed in the mastoid cells, but no pus or granulation tissue.

Microscopically, slight halisteresis was demonstrated besides granular decay of the bone and fully developed bony necrosis. The covering membranes (the "endosteum") were thickened, showing few cells and

22. Larsen, B.: Nord. med. **33**:685, 1947.

vessels, hyalinized; there were no remnants of granulation tissue or chronic round cell infiltrates. Similar changes were not found in the control material.

The facial canal was macroscopically completely soft in 18 patients, or 36 per cent, whereas in the rest of the patients it was hard like ivory. In the microscopic examination granular bony decay was found, and the covering membranes were thickened.

The Facial Nerve.—In 28 patients, or 56 per cent, the nerve was unquestionably edematous macroscopically, in 2 possibly atrophic and in 1 the changes were so violent that the continuity of the nerve was completely interrupted. In the latter case the microscopic examination revealed edema of the nerve trunk, fibrinous exudation, fresh hemorrhage and marked degeneration of axis-cylinders and medullary sheaths. The findings in the the nerve sheath were slight "cicatricial" alterations, slight sclerosis, normal nerve branches and a small vein containing organized remnants of a thrombus. Between nerve and nerve sheath an accumulation of fluid could be observed, consisting of edema and fibrin.

In 20 per cent of the cases of Bell's palsy bony decay or fully developed necrosis was thus demonstrated in the cells of the mastoid process, and in 36 per cent, in the wall of the facial canal. In more than 50 per cent of the cases the nerve was edematous, and in the only case in which microscopic examination was possible exudative changes of of the nerve sheath and, in addition, pure degenerations of the nerve proper were demonstrated.

ETIOLOGY AND PATHOGENESIS

Two theories have been advanced to explain the pathology of Bell's palsy, both of them by French authors. According to the first, Bell's palsy is due to an inflammation, a latent (masked) nonsuppurative otitis media being the responsible factor; according to the second theory, Bell's palsy is due to an aseptic disturbance of the blood supply.

A number of observations seem to indicate that the genesis of the disease must be explained as a disorder of the vasa nervorum. Audibert, Mattei and Paganelli [23] expressed the belief that when a patient gets a sudden massive attack of peripheral facial palsy, after suffering, perhaps, from a half a day's or a day's pain behind the ear but otherwise displaying no abnormal symptoms, it can be explained in one way only: The vascular supply of the nerve has been blocked. (The facial nerve gets its blood supply partly through a branch of the middle meningeal artery, partly from the internal auditory and lastly from the stylomastoid artery.) In

23. Audibert, V.; Mattei, C., and Paganelli, A.: La paralysie faciale périphérique dite "à frigore" est fonction d'une atteinte artérielle des vasa-nervorum, Presse méd. **44**:1049, 1936.

an extremely interesting work these authors have rendered the theory of a failure of the blood supply as the cause of Bell's palsy probable, and they have maintained: *La paralysie faciale périphérique dite "à frigore" est fonction d'une atteinte artérielle des vasa-nervorum* (The peripheral facial paralysis called "à frigore" is an effect of an arterial disturbance of the vasa nervorum). They do not use the expression *une lesion,* as this would imply a serious prognosis and therefore could not be applied to the slight and transient type of paresis, but they use the term *atteinte* (slight injury).

The fact that in some patients the facial nerve degenerates completely, with ensuing permanent paresis, proves that the nerve has been vitally affected. The same authors criticize the term "e frigore" and ask, "When a patient gets a hemiplegia or a hemiparesis, do we then speak of a paresis 'à froid'? We don't, but we know it is a case of spasm, thrombosis or emollition, all of them dependent on a disturbance of the blood supply." Continuing, they expressed the view that exactly the same applies to the facial nerve: A block of the vessels of the nerve is the only way in which the suddenly occurring facial palsy can be explained. If the block does not set on suddenly, the blood supply failing only gradually, the paresis develops little by little.

This discordance of opinions may, I suppose, be only apparent, as nothing excludes the possibility that the cooling (*à frigore*) acts on the vessels of the nerve, disturbing the blood supply.

A series of observations renders a disturbance of circulation probable. According to the theory of Mygind and Dederding,[24] which has gradually been universally accepted, Ménière's disease is due to an edema of the labyrinth which is dependent on failure of the vasomotor function. Many of the patients suffering from Bell's palsy display the same peculiarities as patients with Ménière's disease, such as hardness of hearing, buzzing of the ears, vertigo, nystagmus, slight pleocytosis and, possibly, edema of the mastoid process and along the linea nuchae. The authors then pointed out that, just as the symptoms in Ménière's disease are caused by edema arising in the narrow, inelastic labyrinth, so Bell's palsy is produced by edema of the facial nerve arising in the narrow, inelastic fallopian canal. This view was confirmed by the fact that in several cases they had seen an improvement of the paresis on administration of a powerful diuretic.

Pollak,[25] too, believes that a disturbance of circulation is the cause of the paresis, an idea which will be dealt with presently.

24. Mygind, S. H., and Dederding, D.: Bidrag til de "rheumatiske" facialisparesers pathogenese cg behandling, Dansk otolaryng. selsk. forhandl., 1929, p. 12.

25. Pollak, E.: Die Beteiligung des nervösen Apparates an den Erkrankungen des äusseren und mittleren Ohres, in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1924, vol. 2, pt. 1.

Del Piano, Bergara and Bergara [26] also expressed the belief that Bell's palsy is due to a disturbance of circulation occurring in the fallopian canal, by which the nerve is compressed. They referred to the fact that the nerve is supplied with blood from several vessels and suggested that the convergence of these arterial streams which flow in different directions might produce a condition of stasis that would exert pressure on the nerve. The facial palsy often develops in the night, for which reason these authors belileved that the horizontal position may be thought to favor its occurence.

Ballance and Dued Tickle,[28] Collier and Morris have expressed the belief that primarily the nerve becomes edematous and swollen and consequently is compressed in the fallopian canal, this compression being the real cause of the paralysis. I do not think this is correct. In all probability the nerve is paralyzed owing to primary ischemia, and consequently the nerve becomes edematous and is now, secondarily, compressed in the fallopian canal.

This doubt of mine is strongly supported by investigations carried out by Lewis, Pickering and Rotschild,[29] who studied the paralysis produced by applying pressure to human limbs with the sphygmomanometer cuff and by applying localized pressure on single nerves and showed that the paralysis was due not to the pressure on the nerves but to local ischemia.

Grundfest [30] succeeded in eliminating the factor of anoxia by immersing a frog nerve in oxygenated oil totally enclosed within a pressure chamber. He found that a pressure of 1,000 atmospheres was necessary to abolish conduction in the nerve. In the production of clinical paralysis, pressures of a much less degree are effective over a period of minutes or hours, the part played by cessation of circulation thus becoming the chief causative factor.

Further investigations, carried out by Denny-Brown and Brenner,[31] expressed the same view: "The effect of pressure on the nerve is considered to be due entirely to ischemia."

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26. del Piano, J. I.; Bergara, C., and Bergara, R.: Die sogenannten Facialislähmungen "a frigore," Zentralbl. f. Hals-, Nasen- u. Ohrenh. **25**:418, 1936.

27. Footnote deleted by the author.

28. Tickle, T. G.: Nerve Graft Operation for Facial Paralysis, Laryngoscope **49**:475, 1939; Surgery of the Facial Nerve in Three Hundred Operated Cases, Laryngoscope **55**:191, 1945.

29. Lewis, T.; Pickering, G. W., and Rotschild, P.: Centripetal Paralysis Arising Out of Arrested Bloodflow to the Limb, Including Notes on a Form of Tingling, Heart **16**:1, 1933.

30. Grundfest, cited by Lewis, Pickering and Rotschild.[29]

31. Denny-Brown, D., and Brenner, C.: Lesion in Peripheral Nerve Resulting from Compression by Spring Clip, Arch. Neurol. & Psychiat. **52**:1 (July) 1944; Paralysis of Nerve Induced by Direct Pressure and by Tourniquet, ibid. **51**:1 (Jan.) 1944.

Attention may also be drawn to a paper by Brain, Wright and Wilkinson [32] on the spontaneous compression of both median nerves in the carpal tunnel which lies between the tough transverse carpal ligament and the bones of the wrist joint. They concluded that the paresis "like all pressure lesions of nerves" was due to ischemia. The reaction of the nerve to pressure was edema, which increased the pressure still further and so led to a vicious circle.

There is no little resemblance between the median nerve and accompanying vessels in the inelastic carpal tunnel and the facial nerve in the fallopian canal.

All these investigations, especially those carried out by Grundfest, tend to show that it is highly improbable that the paralysis should be due to a compression of the nerve in the fallopian canal. The rather discouraging results of decompression at the early stage of the disease, as will be shown later, speak in favor of this view.

My systematic pathologic-anatomic investigations also point to a vascular disorder. It will be remembered that in 20 per cent of the cases osseous alterations were found in the mastoid cells, especially around the stylomastoid foramen, and in 36 per cent, in the wall of the facial canal. Microscopically, bony decay and bony necrosis and in some cases exudation were found, but not in a single case granulation tissue or round cell inflammation. In the few cases in which bacteriologic investigations were carried out, the culture was negative.

And what did earlier casuistic reports tell?

Chéridjian in his second case found that the antrum was surrounded by a zone with profuse hemorrhage (*une état local très fortement congestif*) near the upper bend of the nerve, but not pus and no granulations. Flodgren near the facial canal found three large cells containing a reddish brown, viscous, semisolid mass "quite obviously stale blood," and Cawthorne in the nerve near the stylomastoid foramen found several tiny hemorrhagic streaks running longitudinally.

These findings, too, must most probably be explained as a disorder of the circulation, and the same applies to the nerve itself: In the few cases in which microscopic investigations of the nerve were carried out, edema and pure degenerative changes were observed, but no signs of inflammation.

Everything thus seems to indicate that in Bell's palsy a "dysregulation" of the circulation is the primary and central feature of the pathogenesis.

The consequences of a lack of blood supply of the nerve becomes, as stated by Pollak, a *Quellung* of the nerve, an edema with subsequent

32. Brain, W. R.; Wright, A. D., and Wilkinson, M.: Spontaneous Compression of Both Median Nerves in the Carpal Tunnel, Lancet **1**:277. 1937.

degenerative changes; the nerve is thus compressed in its inelastic bony canal, which causes a further impairment of the vascular supply, so that a vicious circle arises. The process is reversible, as the changes subside if the normal blood supply is soon reestablished.

Besides preventing the centrifugal impulses from getting on, which results in paralysis of the musculature, the lesion of the nerve also blocks the centripetal tonus arising from the periphery, with resulting inactivity and atrophy of the facial nuclei, which, in turn, involves descending degeneration of the entire peripheral extension of the neuron. Depending on the size of the vessel that is blocked and the rapidity and the degree to which it takes place, the paresis develops suddenly or gradually, and becomes massive or partial, and it depends on the rapidity with which the blood supply of the nerve is reestablished whether irreparable changes occur—in other words, whether the patient recovers his lost mobility wholly or in part.

In some cases the nutrition of the wall of the fallopian canal and of the mastoid cells is also disturbed, which causes bony decay or fully developed bony necrosis, a reaction collateral to the changes of the nerve itself.

What the cause of this "dysregulation" is and how the mechanism is released I do not know. Especially French authors, Lannois,[33] Mackensie,[33] Deneau,[33] Roche [33] and others have for a number of years maintained that in many cases of Bell's palsy there is an underlying nonsuppurative otitis media. Escat [34] even expressed the opinion that in all cases Bell's palsy is due to atypical otitis, even when every symptom of acute otitis media is lacking, when no specific infection (syphilis, herpes zoster) can be demonstrated. In the opinion of this author it is a periostitis of the fallopian canal, an osteitis, a cellulitis serofibrinosa, an antritis serofibrinosa, an edema of the neurilemma or in severe cases a genuine neuritis.

Barraud is of the same opinion:

. . . Je crois qu'une bonne partie et même je serais porté par mon experience personnelle à croire que la plus grande parties des paralysies de nerf facial "à frigore" sont dues à des otites atypiques avec congestion, inflammation ou même infection de certains groupes de cellules mastoidiennes avoisinant le trajet du facial ou de peri-nevrites de nerf lui-même: dans les deux cas la paralysie est due à une compression du nerf dans la canal de Fallope, rigide et étroit (I believe that a part, and I would be inclined by my personal experience to believe that even the greatest majority, of the paralyses of the facial nerve "à frigore" are due to atypical otitides with congestion, inflammation or even infection of certain groups of mastoid cells adjacent to the course of the facial nerve, or to perineuritis of the nerve itself: in the two cases the paralysis is due to a compression of the nerve in the rigid and narrow fallopian canal).

33. Cited by Barraud.[18]

34. Escat, E.: Traitement chirurgical de la paralysie faciale dite "à frigore," Rev. de stomatol. **30**:614, 1928.

He continues:

. . . L'origine otique de cette paralysie explique très bien les symtomes qui l'accompagnent et qui seraint difficiles à expliquer en dehors d'elle surtout les douleurs qui precèdent la paralysie et qui siégant dans l'oreille, sur l'apophyse mastoïde et qui s'irradient dans la face et le cou comme celles des otites (The otic origin of this paralysis explains very well the symptoms which accompany it and which would be difficult to explain in any other way, especially the pains which precede the paralysis and which are located in the ear or the mastoid apophysis and which radiate to the face and the neck as those of otitides.)

His view on the pathologic nature of the disorder then follows:

. . . J'estime que cette paralysie périphérique est toujours causée par une compression du nerf dans le canal de Fallope est une maladie otique par conséquent justiciable de l'intervention opératoire d'un otologiste (I believe that this peripheral paralysis is always caused by a compression of the nerve in the fallopian canal and consequently is an otic disorder amenable to the operative intervention of an otologist.)

This point of view is supported by the pathologic changes of the drum which in some cases may be observed, the acousticovestibular symptoms which may take place, and the pains.

That the drum is red is no proof of an underlying otitis. If the theory of a disturbance of circulation of the vasa nervorum is accepted as the primary and central feature of the pathogenesis of the palsy nothing may prevent one from explaining the color changes of the drum in the same way, as a simultaneous, collateral reaction to the same noxa.

The acousticovestibular symptoms may of course be due to an underlying latent otitis media, which irritates the labyrinth, but they may, too, and I think more naturally, be explained in the same way as they are in Ménière's disease, as a result of a disturbance of the circulation, just as the palsy itself is supposed to arise.

As to the pains, they may be localized to the ear proper, but more often they are found behind the ear. The cause is unknown, but edema of the firm tissues comes into question. They may, on the other hand, be due to a vascular spasm.

Summarizing the symptoms: the pains, the pathologic changes of the drum and the acousticovestibular findings do not need an underlying otitis media in order to be explained; they may all of them, and I think more satisfactorily, be explained as simultaneous, collateral reactions to a disturbance of circulation, arising just as the facial palsy itself.

Many other observations speak against latent otitis media as an underlying factor. In cases of a manifest infection of the tympanic cavity (acute suppurative otitis media) and not a supposed infection (as is described by some authors in Bell's palsy) facial palsy is found in only 0.5 per cent of the patients, and here the rate must be supposed to be much greater if a middle ear infection is the underlying cause.

Further, if latent otitis media really is responsible for Bell's palsy, there ought to be transitions from the latent otitis to the manifest middle ear suppurations accompanied by facial palsy, and this is definitely not the case.

Last, but not least, the microscopic investigations differed profoundly in cases of Bell's palsy, in which pure necrosis but no sign of inflammation was found, and in cases of mastoiditis, in which round cell infiltration and granulation tissue always could be demonstrated.

From these different points of view it must be concluded that the resemblance between otitis media and Bell's palsy is only apparent and that a latent infection of the middle ear cannot be accepted as explaining the majority of cases of Bell's palsy.

What releases the mechanism of the disturbance of the vasa nervorum I do not know, as already stated. So many patients have been exposed to cold just before the palsy arose that this moment cannot be neglected, but how the cooling works is not clear, even if a vascular spasm may be suspected.

Further, Panneton [35] has described how a facial palsy occurred in connection with extraction of a tooth, the paresis being supposed to be reflex released, and I have observed a similar case myself.

Lastly, Arrieta [36] and Bergonzi [37] have reported cases in which peripheral facial palsy occurred as a consequence of a sudden frightening; the palsy could not be attributed to hysteria, and the authors mentioned as a possibility that, owing to a sudden increase of the blood pressure, a hemorrhage had occurred in the facial canal, causing the nerve to be compressed.

Bell's palsy has become a collective diagnosis for all peripheral facial pareses, in which it has been impossible to demonstrate any local causation.

No doubt many cases may be explained by a more thorough examination; a lumbar puncture for instance will occasionally reveal an intracranial lesion which could not be diagnosed without a puncture.

But from my point of view the question narrows down to this:

Bell's palsy is a pathogenetic entity, the primary and central feature being a disorder, a "dysregulation" of the circulation, in most cases affecting only the nutrition of the nerve as the most susceptible tissue, from which an ischemic paralysis arises, in others affecting also the

35. Panneton, P.: Tentative d'explication des paralysies faciales périphériques improprement appelées "à frigore," Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:639, 1938.

36. Arrieta, A.: Sur un cas de paralysie faciale d'origine emotine, Rev. de laryng. **46**:599, 1925.

37. Bergonzi, M.: Paralisi del faciale "ex emotine," Zentralbl. f. Hals-, Nasen- u. Ohrenh. **35**:479, 1943.

nutrition of the facial canal and the mastoid cells, causing bony necrosis. As in 6 of my cases the bony necrosis was highly developed around the stylomastoid foramen, whereas the rest of the mastoid cells were normal, and as in the other 5, in which necrosis could be demonstrated in the entire cellular system, the changes were much more pronounced around the stylomastoid foramen, it must be suspected that the vascular block takes place near the latter.

The rapidity with which the paralysis begins, the degree to which it develops, the time it lasts and the degree of the pathologic changes which may be noted in the nerve and the osseous structures all depend on the size of the vessel that is blocked and on the rapidity and the degree to which the circulation is cut off and, eventually, reestablished.

The cause may vary, and at present not much is known about it, but the pathogenesis is in all probability the same: a primary ischemic paresis of vascular determination.

II

SURGICAL THERAPY

As experience has shown that in the great majority of cases (according to Tickle, in 85 to 90 per cent) Bell's palsy subsides under suitable conservative treatment, and as Ballance and Duel introduced the decompressive treatment, the important question immediately arose how to try to select for operation the 10 to 15 per cent of patients who do not obtain spontaneous recovery.

In a number of publications Ballance and Duel insisted on the faradic reaction as indicator, maintaining that if this reaction becomes negative and the patient is treated conservatively, he either will not recover at all or will recover only partly. This line was followed by Tickle, Morris and Collier, but no material has been produced to elucidate the matter.

Does the faradic reaction actually give reliable information about the prognosis? By no means.

The following case record may be given as an example of complete spontaneous restitution, a negative faradic response.

A woman aged 68, who had never had suppurative otitis media, suddenly experienced massive right-sided Bell's palsy three days before hospitalization. This was not accompanied by acoustic phenomena or herpetic eruption, and her temperature was normal. There was slight dizziness. Both tympanic membranes were calcified, and there was slight reddening along the manubrium of the right malleus. Whispering was heard at 5 feet (1.5 meters) in both ears. The audiogram showed decreased acuteness of hearing of the tone-perceiving type, corresponding to her age. There was spontaneous horizontal rotatory nystagmus to the left (rotation $5 \times \frac{45'' - 37''}{22'' - 26''}$). The blood pressure was 205 systolic and 105 diastolic. The remaining objective examinations, including the neurologic examination, roentgen study of the ears and ophthalmoscopy, showed normal conditions. The spinal

fluid cell count was 0/3; the albumin content was 13 mg. and the globulin content 2 mg. per hundred cubic centimeters. The faradic reaction was negative. Sixteen days after, the first signs of recurring mobility were observed, and three and one-half months later the paresis had subsided completely (fig. 3).

Another instance may be given:

A radical operation was performed on a man aged 34 for cholesteatomatous otitis media. Five days after the operation the first signs of facial palsy appeared. For this reason the tamponade was removed. Two days later, the paresis was massive, and the faradic reaction was negative. The condition remained stationary for three weeks; then the facial palsy began to subside, and four months afterward the only remnant of the paresis was slight synkinesis.

Thus it is seen that the negative faradic reaction by no means excludes a complete spontaneous restitution.

Fig. 3.—Bell's palsy. In spite of a negative faradic reaction the patient regained spontaneously full mobility in two and one-half months.

This is also readily understood: When a muscle is paralyzed, its electric excitability immediately increases; i. e. the chronaxia of the muscle is shortened, which means that a stimulus of shorter duration is capable of exciting contractions. After a short period of time, however, the excitability of the muscle decreases; in other words its chronaxia is extended, so that a stimulus of longer duration is required to excite contractions. The faradic current is a high frequency alternating current, and the fact that the faradic reaction becomes negative just means that the stimulus is inadequate because it is of too short duration to excite contractions of the musculature, the chronaxia of which has become extended. Therefore, no safe conclusions as to the prognosis of the paresis can be drawn from a negative faradic reaction. I have repeat-

edly emphasized this fact, and Violé [38] and Rasmussen,[39] too, have had the same experience. Of more value is the positive faradic reaction, but even this response does not guarantee a complete spontaneous recovery.

Electromyographic examinations may afford better information, as suggested by Feinstein,[40] but until this is forthcoming one must try to approach the question of indications empirically.

In the presence of otogenous paresis Ballance and Duel recommend immediate decompression, but I have shown empirically (Kettel, 1943) that this indication is not tenable for all cases, and in a more recent publication Riskær [41] admitted that I am right.

I have now tried the same procedure in cases of Bell's palsy. As in all the patients with otogenous facial palsy in whom the condition subsided spontaneously and completely, the first signs of beginning mobility were observed before two months had passed (Kettel, 1943) and as exactly the same observation was made in patients suffering from Bell's palsy (Arnvig [42]), I have fixed the expiration of this period as the time limit for conservative treatment of Bell's palsy.

Morris stated that six to eight weeks was the utmost limit for expectant treatment of Bell's palsy which shows no signs of beginning mobility or only slight signs, but he added that if the faradic reaction becomes negative or the reaction of degeneration becomes positive before this period has passed, the decompression must be carried out as soon as possible. Tickle (1939) wrote: "In my experience the 85 per cent that recover had never lost their response to faradic stimulation, ... the 15 per cent who lose their response to faradic stimulation and show no improvement in from six to eight weeks should have a decompression of the facial nerve."

At the risk of being regarded as immodest, I must say a few words about the results obtained by me in the use of the Ballance-Duel and Bunnell [43] operation, in other, and technically far more difficult, cases of peripheral facial palsy; otherwise, I might justly be asked whether the less favorable results I have had in Bell's palsy were not due to a faulty technic.

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38. Violé, P.: Experiences in Surgery of Facial Nerve, Laryngoscope **54**:455, 1944.

39. Rasmussen, H.: Den otogene facialisparese med saerligt henblik paa behandlingen, Nord. med. (Hospitalstid.) **28**:2214, 1945.

40. Feinstein, B.: The Application of Electromyography to Affections of the Facial and the Intrinsic Laryngeal Muscles, Proc. Roy. Soc. Med. **39**:817 (Oct.) 1946.

41. Riskær, N.: The Course of Otitic Facial Palsy Under Adequate Treatment of Disease of the Ear, Acta oto-laryng. **34**:280, 1946.

42. Arnvig, J.: Personal communication to the author.

43. Bunnell, S.: Surgical Repair of the Facial Nerve, Arch. Otolaryng. **25**:235 (March) 1937.

I have made 50 decompressions for otogenous paresis, and all the patients, whose facial palsy was massive, have recovered their mobility partly or in full. Moreover, I have made nerve graftings, or nerve sutures, in 50 cases in all; 10 patients were operated on recently, so that the results cannot yet be estimated, but 36 of 40 patients, or 90 per cent, whose period of observation has elapsed recovered mimicry. Among these there are many in whom the proximal end of the graft had to be united with the end of the facial nerve just distad to the geniculate ganglion, and several in whom the distal end of the facial nerve had to be sought in the parotid gland. If the causes of the failure of operation in the patients are analyzed, it will be seen that the first patient had tuberculosis of the middle ear; the next had partial muscular atrophy, and a bad prognosis was made before operation, but the patient insisted on operation; the third patient, a woman aged 72, recovered no mobility in spite of a perfect grafting; in the last patient the nerve was only partly ruptured, but in spite of this fact and the fact that a transplant was grafted the operation failed.

As stated, these operations required far more technically than simple decompression, but when I include all cases, even the one in which the grafting should have been avoided, owing to the poor condition of the musculature, I have still obtained a percentage of recovery of 90; therefore I believe I may venture to maintain that the less satisfactory results which I have obtained by means of decompression in cases of Bell's palsy cannot be attributed to a failing technic.

The first of the three indications for surgical treatment of patients suffering from Bell's palsy was formulated by me as follows:

1. *A decompression should be made in cases in which signs of beginning mobility have not appeared after a period of observation of two months.*

I have operated on this indication in 25 cases. In all the paresis was massive and the faradic reaction negative. The results are stated in the table.

Roughly speaking, the facial nerve divides into a superior, an intermediate and an inferior branch, innervating respectively the musculature of the forehead, that around the eye and that around the mouth. Each of the nerve branches innervates different groups of muscles, but with a view to perspicuity the results stated in the table express the effects of the first, second and third branches on the frontalis (wrinkling the forehead), the orbicularis oculi (closing the eye) and the zygomaticus (smiling and laughing) muscle. A more detailed analysis follows later.

The degree of mobility is stated by means of the figures 0 to 4; it goes without saying that this grading conveys the facts only approximately.

0 = no mobility (0% of the normal)
1 = slight mobility (about 25% of the normal)
2 = greater mobility (about 50% of the normal)
3 = vigorous mobility (about 75% of the normal)
4 = full mobility (about 100% of the normal)

What this means in practice is best seen from figure 4, illustrating conditions in the case of the mouth.

Examinations after operation were made, of course, at a time when further improvement had ceased. Only 20 of the 25 patients operated on are included in the statement; in 2 of the remaining ones the continuity of the nerve was interrupted (cases 4 and 5, papes 435 and 436), which rendered a nerve grafting, or approximation of the ends, necessary, for which reason the results must be estimated from a different angle, although both of the patients recovered their mobility. The last 3 patients were recently operated on, so that the results cannot yet be estimated.

A look at the table shows that: (*a*) The paresis remained unnoticed in all the patients when the face was at rest; in 3 of them, however, a slight difference between the opening of the palpebral fissures was seen. (*b*) All of the patients were able to close their eyes and to close them tightly. (*c*) In all the patients synkinesis varying from slight to more severe could be demonstrated.

On closer analysis it appears now that only 2 patients (table, and figs. 5 and 4 *D*), or 10 per cent, had recovered complete mobility in the entire part innervated by the facial nerve; they had been operated on two and three months, respectively, after the onset of the paresis.

Practically all the patients had noticed the first signs of returning mobility around the mouth, preceded by slight paresthesia and a sensation of increased tonus. On examination of the function of the groups of muscles innervated by the first, second and third branches of the facial nerve, respectively, it may be seen that there is a difference of degree. If, as a mere supposition, the grades (from 0 to 4) that have been given to all the patients for the mobility they have obtained in the frontalis muscle (first branch) are added and the sum divided by the number of patients, and if the same calculation is then made for the orbicularis oculi (second branch) and the zygomaticus (third branch), three quotients will result, namely 1.8, 2.8 and 2.6, respectively. It must be frankly admitted that this is a rough calculation, as some inaccuracy in the grading cannot be avoided. Still, it may be seen that the musculature of the forehead displays the poorest regeneration and that the conditions are fairly identical in regard to the orbicularis oculi and the zygomaticus.

As already mentioned, the values stated in the table for the third branch refer to the action of the zygomaticus (plus the risorius), the

muscles on which smiling and laughing depend. On examination of the function of the remaining muscles acting on the mouth it will be seen that, while the contraction of the orbicularis oris was comparatively good, 15 of 20 patients were either completely unable to raise the upper lip

Fig. 4.—Different degrees of mobility of the mouth regained. *A*, slight (25 per cent). *B*, more pronounced (50 per cent). *C*, strong (75 per cent). *D*, full mobility (100 per cent).

In the table the grades are 1, 2, 3 and 4, respectively.

and wrinkle the nose or were able to do so only slightly (quadratus labii superioris plus caninus muscle), 16 of 20 were unable to draw the lower lip downward and laterad (quadratus labii inferioris plus triangularis muscle), and about half the number were unable to whistle (incisivi muscles). Exactly the same picture is seen in patients suffering from otogenous paresis, who have either had a nerve grafting or have spontaneously recovered partial mobility if the continuity of the nerve has

Fig. 5.—Bell's palsy of two months' duration. After decompression the patient regained full mobility, as shown in figure 4 *D*.

not been interrupted. Only 3 cases are known (Martin,[44] Kettel, 1943) in which the frontalis muscle recovered its function after a nerve grafting.

It is difficult to give an explanation of this fact. Martin surmised that the musculature of the forehead contains the fewest contractile elements and that the latter degenerate before the musculature can receive nervous impulses again.

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44. Martin, R. C.: Recent Experiences with Operation on Facial Nerve, Arch. Otolaryng. **32**:1071 (Dec.) 1940.

Results Recorded on Reexamination of 20 Patients on Whom Decompression of the Facial Nerve Had Been Done for Bell's Palsy

Patient	Age, Yr.	Sex	Duration of Palsy Till Day of Operation, Mo.	Destruction of Mastoid Cells	Destruction of Wall of Facial Canal	Degree of Edema of Facial Nerve	Palsy When Face Is at Rest	Degree to Which Forehead Can Be Wrinkled	Degree to Which Eye Can Be Screwed Up	Epiphora	Degree to Which Patient Can Smile and Laugh	Degree to Which Upper Lip Can Be Raised	Degree to Which Lower Lip Can Be Lowered	Ability to Whistle	Synkineses	Final Result *		
																I	II	III
																M. Frontalis	M. Orbicularis Oculi	M. Zygomaticus and M. Risorius
1	19	F	2	0	0	0	0	4	4	0	4	4	4	+	+	4	4	4
2	11	M	1	0	0	+++	0	0	3	(+)	2	0	0	+	+	0	3	2
3	55	F	3	0	0	+++	0	4	4	0	4	4	4	+	+	4	4	4
4	42	F	1	0	0	++	0	4	4	(+)	3	0	0	0	+	4	4	3
5	48	M	2	0	0	+	0	3	4	0	3	3	0	+	+	3	4	3
6	2	M	3½	0	0	0	0	2	1	+	3	0	0	0	+	2	1	3
7	40	M	5	0	0	++	0	2	3	(+)	3	0	0	0	+	2	3	3
8	36	F	2½	0	0	Atrophic	0	1	4	0	2	0	0	0	+	1	4	2
9	29	F	2	+	0	++	0	4	3	0	3	2	0	+	+	4	3	3
10	67	M	2	0	0	0	0	1	3	(+)	3	0	0	0	+	1	3	3
11	29	M	2	0	+	++	0	3	3	0	2	1	2	+	+	3	3	2
12	31	F	2	0	0	+++	0	0	2	+	2	0	0	0	+	0	2	2
13	32	F	1½	0	+	+	0	3	3	+	2	0	0	(+)	+	1	3	3
14	32	M	1½	0	+	+++	0	1	3	0	2	0	0	(+)	+	0	3	2
15	45	F	2	0	0	+	0	(1)	2	+	2	0	0	+	+	(1)	2	2
16	46	F	2½	+	0	++++	0	(1)	3	0	2	0	0	0	+	(1)	3	2
17	34	M	7/30	0	0	++	0	0	1	+	2	0	0	0	+	0	1	2
18	13	F	9/30	0	0	+++	0	1	3	0	3	2	2	+	+	1	3	3
19	34	M	2	0	+	++	0	4	3	+	2	0	0	+	+	4	3	2
20	37	F	12/30	0	0	+++	0	0	1	+	1	0	0	0	+	0	1	1

* As to the degree of mobility, compare with figure 4.

Why have better results not been obtained? One might question oneself: Was the decompression made too late, and would better results have been obtained if decompression had been made at once? To find an answer to these questions, 3 patients were operated on a few days after the onset of the paresis:

A woman aged 37 was admitted on Dec. 5, 1945. She had never had any suppurative otitis media. Eleven years before admission she underwent massive left-sided Bell's palsy, which subsided spontaneously and completely in a couple of months. Twelve days before admission massive Bell's palsy developed on the right side, accompanied by pain behind the ear, gustatory disturbances and hyperacusis. There were no vestibular symptoms, no eruption of herpes zoster and no increase of temperature. Otoscopy, acousticovestibular examination, ophthalmoscopy, the

Fig. 6.—In spite of decompression done twelve days after the onset of the palsy, the patient recovered only partially. *A*, before operation. *B*, seven months later.

remainder of the neurologic examination and of the general physical examination, and roentgen study of the ears showed nothing abnormal. The spinal fluid cell count was 2/3; the albumin content was 10 mg. and the globulin content 1 mg. per hundred cubic centimeters.

A decompression of the right facial nerve was made at once, with the following observations being recorded: "The cellular system is completely normal, with large glossy cells; the facial canal is perhaps slightly softer than the normal. Nothing happens at once as the nerve sheath is slit open, but in the course of a few minutes the nerve bulges high, being pressed through the slit like a whitish balloon."

Microscopic examination of cells and tissue from the facial canal showed nothing unquestionably pathologic.

The mobility was returning after a course of about two months, and the improvement continued for another two months, but only to a slight degree, and

examination seven months after the operation showed that the patient's condition had remained stationary without further progress for three months; the paresis was not to be seen at rest, but there was only a suspicion of mobility in the parts innervated by the first and third branches, whereas the eye could close, and close tightly, though only with slight strength (fig. 6).

A man aged 34 was admitted on Nov. 6, 1945. He had never had suppurative otitis media. Seven days before admission massive left-sided Bell's palsy developed suddenly, with pain behind the ear but no acousticovestibular symptoms, eruption of herpes zoster or increase of temperature. Otoscopy, acousticovestibular examination, ophthalmoscopy, the remainder of the general (especially the neurologic) examination, and roentgen study of the ears showed normal conditions. The spinal

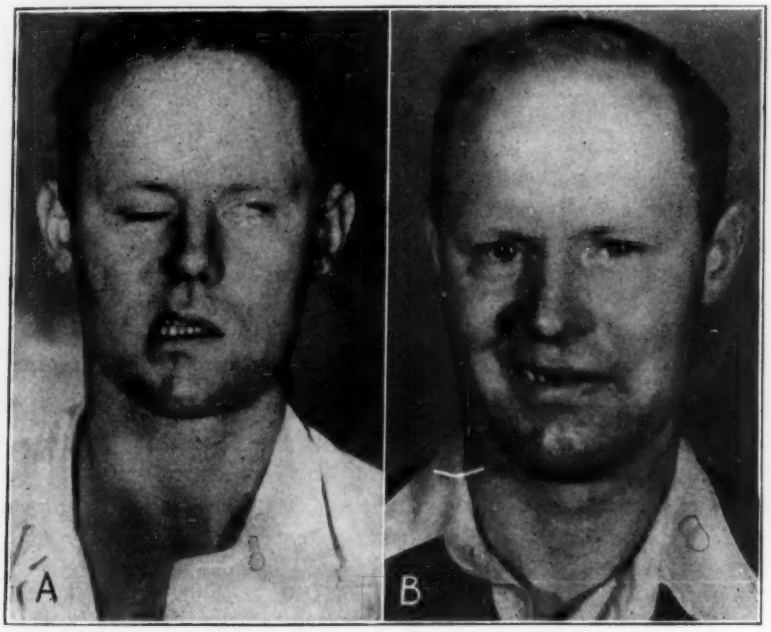

Fig. 7.—In spite of decompression done seven days after the onset of the palsy, the patient recovered only partially. *A*, before operation. *B*, eight months later.

fluid cell count was 0/3; the albumin content was < 10 mg. and the globulin content < 1 mg. per hundred cubic centimeters.

Decompression of the left facial nerve was performed at once, and the following note was made: "The cells are completely normal everywhere; the bone, spongy. When the nerve sheath is slit open, the nerve bulges distinctly, which is especially conspicuous after a few minutes' observation."

Signs of returning mobility were not noted until six months after the operation, and full mobility was not regained. The patient can wrinkle his forehead and can close his left eye but cannot screw it up to the same degree as the one on the other side. He can only partially smile and laugh (fig. 7).

In a third patient, a girl aged 13, the result was similar. She was operated on eight days after the beginning of the palsy.

In these cases decompression was performed on the seventh, eighth and twelfth days after the onset of the disease, respectively; in all of them great edema of the nerve was found, but in spite of the fact that the pressure was thus soon obviated, the clinical result is not encouraging. It may be added that the operations proceeded smoothly without causing any trauma whatever to the nerve.

It will appear from the cases reported here that even if a decompression is made immediately after the onset of the paresis, this is by no means a guaranty that the patient will recover complete mobility; on the contrary, 2 of the 3 patients mentioned are among those with the poorest mobility.

Is it due to traumatic injury in the course of the operation? No! The operations proceeded quite smoothly, without the least pressure on the nerve, still less any violent trauma except, perhaps, in case 4 (page 435).

Is it the age of the patient that decides the degree of recovered mobility? It is not a factor either. A look at the table shows that one of the patients (no. 3) who recovered the greatest mobility is one of the oldest of the patients; conversely, the result obtained in the youngest patient (no. 6) is one of the poorer, and still the greatest regenerative power might be expected in younger persons. Both of these patients had the same postoperative treatment; so the cause cannot be sought there either.

Is the result dependent on the destruction of the mastoid cells? No. In patient 9 there was a considerable osseous focus, but still she recovered mobility to a great extent; in patient 16 there were similar changes but, contrariwise, she recovered only slight mobility. Then, again, there was nothing abnormal in the bony system, on objective examination, in patient 1 or in patient 20; one recovered complete mobility; in the other patient the result was poor.

Is the ultimate result dependent on whether the nerve was edematous and was relieved of pressure in the decompression? By no means. In patient 16 the nerve was enormously edematous, and the clinical result was poor, whereas—despite the same conditions as far as the nerve was concerned—patient 3 recovered mobility to a great extent. On the other hand, in patients 15 and 13 the nerve was only slightly edematous: one recovered great, the other only slight, mobility.

It thus appears from the foregoing considerations that prognostic conclusions as to the ultimate result cannot be drawn from the patient's age, from the objective condition of the mastoid process or from the degree of edema observed in the nerve. The result is undoubtedly dependent on, first and foremost, the degree and the duration of the dysregulation of the vasa nervorum, which must be supposed to be the underlying cause of the paresis.

How, then, should one deal with patients suffering from Bell's palsy at the initial stage?

From 85 to 90 per cent of the patients recover spontaneously and completely, and this fact alone is sufficient to indicate that to far the greatest extent, though not exclusively, the therapy should be conservative. It is of the utmost importance to the ultimate result that the contractibility of the musculature be maintained until the innervation has been reestablished. For this purpose the myotensor of Clemmesen is unique, being constructed according to the principle that the galvanic impulse can be closely adjusted to the chronaxia of the musculature at the given moment.

However, 10 to 15 per cent remain who, as has already been shown, cannot, by means of the faradic reaction, be separated from those who recover completely and spontaneously. I would suggest that the patient suffering from Bell's palsy who has not noticed any signs of returning mobility after two months' observation should be operated on, a decompression being done for two reasons: First because it has appeared that no patient in whom signs of returning mobility cannot be observed after an observation period of two months obtains complete spontaneous recovery (Arnvig) whereas Cheridjian and Flodgren have observed so signal an effect on the paresis immediately after the intervention that there can be no doubt about the efficacy of the operation in their cases. Second the paresis may be associated with other underlying disorders which sometimes cannot be diagnosed before the operation (e. g., neurinoma, Kettel, 1946).

By using this indication I have obtained the result that all the patients were of completely natural appearance when the face was at rest and that all of them recovered their lost mobility to a greater or less extent, but only 2 recovered it completely. A fact that goes to show that the improvement may be due to the operation is that the mobility began rather soon after the intervention—on an average, one month after. But my material does not supply any decisive proof that the same result would not have been obtained by means of further conservative treatment. To decide this question a comparison is required between far larger groups of patients who have been treated surgically, on one hand, and patients who have been treated conservatively on the other. Indication 1 consequently ought to be regarded as serving temporarily until better means for selecting the early cases for operation have been attained.

It will appear from the foregoing comment that I arrive at much the same conclusion as Tickle and Morris, namely, that the patients must be operated on if there are no signs of beginning mobility after a period of observation of about two months, but my reasons differ. Operation is not indicated because the faradic reaction becomes negative—this is, as I have shown, of no significance—but because it has

been empirically stated that no patient in whom the first signs of returning function where not noted before two months had elapsed regained full mobility spontaneously and because there may be a neurogenous tumor which cannot be diagnosed before the operation.

The question about the value of the decompression is, of course, not solved by ascertaining that the intervention is helpful only in the cases in which an osseous focus can be demonstrated, which is easily understood as the bony necrosis is not the cause of the paralysis but a simultaneous reaction to the same noxa from which the latter arose. I can state, for instance, that in 1 of the 2 patients who recovered complete mobility (table) and in whom the reaction of degeneration was positive I could find nothing abnormal at all in the mastoid cells or in the wall of the facial canal, the nerve, too, being macroscopically normal.

The next indication for operation is as follows:

2. *A decompression should be made if the spontaneously recovered partial mobility has ceased before complete restitution has been obtained.*

As far as I know, it has never been observed that a spontaneous restitution has ceased at a certain time and has then continued after a pause during which no progress could be observed. If, therefore, it can be proved that the improvement continues after the decompression, the value of this intervention has been substantiated. This has been observed by Morris in a number of patients on whom he operated six months to two years after the spontaneous improvement had ceased; in 1, even eight years afterward.

I have operated on 17 patients on the basis of this indication. It is too early to say anything about 3 of the patients, and I have not been able to get in touch with 1 other, because he has been away traveling. In 9 of the remaining 13 patients the operation has brought about an improvement of mobility, whereas in 1 the intervention had a favorable effect in another direction, as will be mentioned presently. In 3 patients the decompression was of no effect, but these patients did not get worse either.

One of the 9 patients recovered complete mobility, as will appear from the following case record:

A woman aged 36 years was admitted on Oct. 25, 1945. She had never had any suppurative otitis media. Two and a half months before admission massive left-sided Bell's palsy suddenly appeared, accompanied by gustatory disturbances but not by pain, acousticovestibular symptoms, herpetic eruption or increase of temperature. The paresis remained massive for four weeks; then followed a period of three weeks' even progress; then for three weeks the condition remained stationary. There was a suspicion of function in the first branch; when the eye was closed, a 2 mm. space was left open, and the eye could not be closed tightly. Bell's phenomenon was observed. The mobility in the part supplied by the third branch was as shown in figure 8. The remainder of the general examination, especially the otologic and neurologic phases, ophthalmoscopy and roentgen study

of the ears showed normal conditions. On decompression the nerve bulged somewhat, but it was doubtful whether the bulging exceeded the physiologic limit. The mobility increased at once after the operation and was completely normal six months afterward (fig. 8).

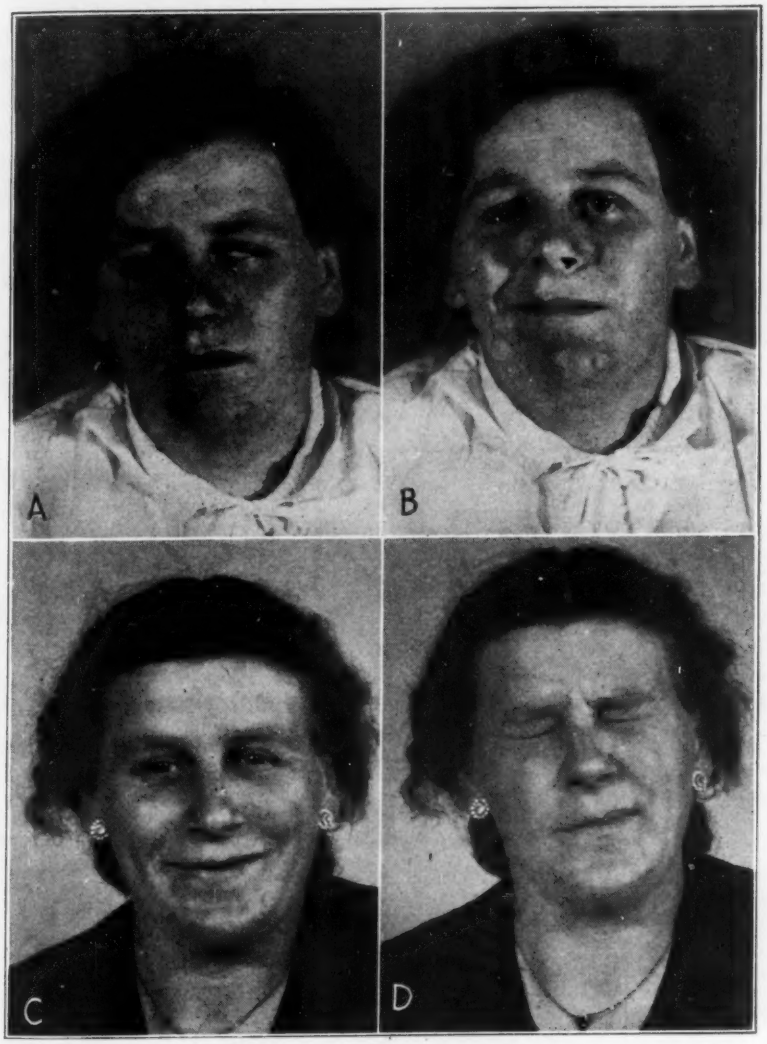

Fig. 8.—*A* and *B*, Bell's palsy of two and one-half months' duration but without improvement during the last three weeks. After decompression, the patient regained full mobility (*C* and *D*).

In the remaining 8 patients a greater or less improvement of the mobility was demonstrated. The paresis had persisted for seven, nine, eleven, twenty-four, thirty-five, thirty-six, sixty and one hundred and twenty months, respectively, and in spite of these long periods the

intervention still produced an effect which is in keeping with the observation cited from Morris.

Degrees of mobility found in postoperative examination of these patients whose paresis healed defectively have not been tabulated, because, when the operations were performed the pareses were partial but had not been graded, so that there is no basis for comparison. The mastoid cells were natural in all the improved patients, but a destruction of the wall of the facial canal was ascertained in 3. In 2, whose paresis had lasted for seven and thirty-five months, respectively, the nerve was distinctly edematous; in 1 with paresis of thirty-six months' duration it was atrophic, whereas it was macroscopically normal in the rest of these patients.

In 1 case operation did not have any effect on the paresis, which had persisted for seventy-two months, but the patient, a 10 year-old child,

Fig. 9.—*A*, Bell's palsy of seven months' duration on the left side, with contracture. *B*, appearance of the patient the day after the operation. The contracture disappeared immediately after decompression without the slightest impairment of the mobility. This tends to prove that the contracture depends on an irritation of the nerve in the fallopian canal (Grindstein's theory) and is not due to shrinking of the muscles.

was relieved of contracture and of the hemicrania, from which she had suffered throughout all these years.

All the patients had a sensation of reanimation of the face, as the tightening sensation had either disappeared or decreased.

The operation was without effect in 3 cases, in which the paresis had lasted seven, eight and a half and twenty-four months, respectively. In one of these the bony system and the nerve were normal, in another a destruction of the wall of the facial canal was ascertained and in the third there were destructions both of the facial canal and the mastoid cells, and the nerve was distinctly edematous (case 8, page 438).

It must be supposed that irreparable injury had already been incurred in the central and peripheral parts of the nerve in the patients who did not improve after the operation.

In addition, two other interesting observations were made in these operations. A distinct contracture was seen in 1 patient before the decompression, but it disappeared immediately after the operation (fig. 9), the patient did not become in the least more paretic than she was before the intervention, and two and one-half years afterward there had been no relapse. I have made the same observation in 2 other patients. This seems to indicate that the view of Grindstein, according to which the contracture is reflex released and due to an irritation of the facial nerve in the fallopian canal and not to a wasting of the musculature, is correct.

This is also in keeping with the fact that these patients stated spontaneously that before the operation they had an unpleasant tightening sensation in the face but that it lessened considerably or disappeared completely after the decompression.

Therefore: If the spontaneous improvement has ceased, a decompression should be made as soon as possible, and when one is faced with a troublesome contracture, this operation should be tried.

Lastly, I have tried decompression in still another group of patients with Bell's palsy on the following indication:

3. *Decompression is indicated in relapsing paresis.*

This group comprises 8 patients who had had facial palsy two to nine times, and not 1 of them has had a relapse since the operation. The period of observation of the first patient is, however, only four and a half years, and even if there has never been an interval between her attacks so long as this, one cannot guarantee that relapse will not occur later. It should, however, be noted that this patient, who was previously troubled by a sensation of masklike features, due to her bilateral facial palsy, has now acquired far less impeded mobility and has been relieved of the troublesome tightening sensation in her face.

By means of the operation three things have been gained in these patients: First, the mobility improved in all of them, and 2 patients recovered completely; second, they have no tightening sensation in the face any more, or only a slight sensation, and, last but not least, they have all of them been relieved of an enormous psychic pressure. How great the latter has been will appear from the fact that 2 of these patients had attempted suicide because of their paresis. It applies to all these patients that previously they had never for a moment felt safe from relapse with all its human and social consequences and that the operation had relieved them of these complexes.

How can it be that even immediate decompression does not secure complete mobility, while it helps and may even completely cure the paresis from which the patient has recovered spontaneously with defect? And why has not one of the patients with relapsing paresis had any attack after the operation?

If it is correct, as Ballance and Duel, Collier and Morris have maintained, that the paresis is due to a compression of the edematous nerve in the fallopian canal, it might be expected that the decompression would restore complete mobility in, at any rate, a great number of the patients and especially in those who are operated on soon after the onset of the paresis. This is not the case, however, and in my opinion the disappointment is due to a misinterpretation of the causation of the paresis.

Morris wrote about the latter, "And here it is essential to give a clear definition of what Bell's palsy is. It is due to inflammation of the nerve sheath or the nerve itself, due to toxaemia or congestion due to changes of temperature occurring in the second portion of the aqueduct whereby nerve impulses are obstructed or totally inhibited."

I agree with Morris that frequently the nerve is swollen, but in my opinion this is a secondary phenomenon.

The primary and central factor is a failure of the blood supply of the nerve owing to a dysregulation of the vasa nervorum. Therefore the nerve is paralyzed. Only secondarily a quellung of the nerve occurs, with the resulting degenerations previously described. When in some cases the decompression, even if performed at the earliest stage of the disease, has not given a satisfactory result, this is due to the fact that in these cases degeneration of the nerve has already taken place.

It seems to me to be probable that the favorable effect of the decompression in many cases is due to the fact that on exposure of the nerve, the circulation of the blood is in some way or other improved by releasing the pressure on the vasa nervorum or perhaps by the establishment of a collateral circulation.

From this point of view it should seem theoretically well founded to make a decompression at once, as soon as the diagnosis of Bell's palsy has been established, in order to improve the circulation as soon as possible. This procedure must, however, be rejected, first, because in 85 to 90 per cent of the patients the paresis subsides spontaneously and, second, because it has appeared that decompression in cases of fresh paresis by no means guarantees 100 per cent recovery. This is possibly due to the fact that some time will pass before the blood passages begin to function, and during this period the nerve is deprived of sufficient blood supply, for which reason greater or smaller peripheral and central degenerations may occur, compromising the ultimate result. Therefore it must also be supposed that in the cases in which the decompression fails to correct recovery with defect, irreparable injury has already occurred in the facial nuclei and in the peripheral neuron.

Failure to obtain an expected effect may also be imagined to be due to the fact that collaterals have not been established as a result of the operation.

The reason why patients with relapsing facial palsy had no relapses after decompression is probably that the circulation was improved.

SUMMARY AND CONCLUSIONS

In 1932 Ballance and Duel began to operate on patients suffering from Bell's palsy, decompressing the facial nerve by opening the fallopian canal from the stylomastoid foramen to the lateral semicircular canal, their view being that the disease is due to edema of the facial nerve, which is compressed in the inelastic bony canal. Ballance and Duel and other Anglo-American authors have by this procedure attained beautiful results.

The aim of the present paper, which is based on 50 cases in which I operated in the manner of Ballance and Duel, represents an attempt (1) to procure a material of consecutive cases in order to estimate the value of the operation—such a publication has been lacking—and (2) to try to arrive at a deeper understanding of the pathology of Bell's palsy.

PATHOLOGIC ANATOMY

Changes which have not been described earlier have been demonstrated both in the mastoid cells and in the facial canal itself.

Cells of the Mastoid Process.—In 11 of 50 cases, or about 20 per cent, there was necrosis of the cells; in 5 cases this spread to the entire cellular system but was especially marked in the apex of the mastoid process; in 1 case the cells were filled with a dark clear fluid. In 6 cases the great part of the cells were normal, but at the apex of the mastoid process and particularly around the stylomastoid foramen a large area of bony necrosis was encountered. The decayed bone could easily be removed by means of a sharp spoon, and after the excochleation the distal part of the mastoid process had been transformed into a deep cavity. In the microscopic examination granular decay of the bone, particularly bony necrosis, was demonstrated, but no round cell infiltration or granulation tissue.

Fallopian Canal.—In 18 patients, or 36 per cent, the fallopian canal was macroscopically completely soft and could be opened in a few minutes, in contrast to the opening of the normally ivory-hard canal, which is a time-consuming work. In 2 cases the bony necrosis was so extensive that the facial nerve had been exposed to view in the fallopian canal. Microscopically, granular decay and fully developed necrosis were observed, but no round cell infiltration and no granulation tissue.

Facial Nerve.—In 28 patients, or 56 per cent, the nerve was unquestionably edematous macroscopically; in 2, possibly atrophic, and in 1 the changes were so violent that the continuity of the nerve was com-

pletely interrupted (case 5). In the latter case the microscopic examination revealed edema of the nerve trunk, fibrinous exudation, fresh hemorrhage and marked degeneration of axis-cylinders and medullary sheaths. In the nerve sheath a small vein containing organized remnants of a thrombus, and between nerve and nerve sheath an accumulation of fluid consisting of edema and fibrin, could be observed.

ETIOLOGY AND PATHOGENESIS

Much seems to indicate that Bell's palsy is a pathogenetic entity, the primary and central feature of which is a "dysregulation" of the circulation, which probably takes place near the stylomastoid foramen. In most cases the nerve as the most susceptible tissue suffers alone; in other cases the surrounding, more resistant bone is also affected. The result is ischemic paralysis and ischemic bony necrosis, respectively. The consequence of the lack of blood supply of the nerve is edema with subsequent degenerative changes; the nerve is thus compressed in its bony canal, which causes further impairment of the vascular supply, so that a vicious circle arises, the process being reversible. Thus the actual cause of the paralysis is the ischemia; the compression in the fallopian canal is only a secondary phenomenon, and not, as maintained by most authors, the real cause of the paralysis. In extreme cases ischemic necrosis of the nerve may be the result, as was seen in case 5.

The rapidity with which the paralysis begins, the degree to which it develops, the time it lasts and the degree of the pathologic changes occurring in the nerve and the surrounding bony system all depend on the size of the vessel that is blocked and the rapidity and the degree to which the circulation is cut off, and, eventually, reestablished.

In many cases Bell's palsy is accompanied by pain in the ear, acousticovestibular symptoms and color changes of the drum, for which reason it has been maintained that in most cases, if not all the palsy is due to an underlying (masked) nonsuppurative otitis media. The said symptoms may more convincingly be explained as vascular phenomena, reactions collateral to the ischemic paralysis and ischemic bony necrosis.

Bell's palsy is in all probability a primary ischemic paresis of vascular determination. The cause may vary, and at present not much is known about it. In many patients the paresis arises after a cooling, and this moment must not be underestimated as a factor which may release the disturbance of the blood supply of the nerve.

SURGICAL THERAPY

In 85 to 90 per cent of the cases Bell's paresis subsides under suitable conservative treatment. In selecting the last 10 to 15 per cent of the patients Anglo-American authors rely on the faradic reaction,

maintaining that if the response becomes negative the patient will not recover at all or will only recover partly if conservative treatment is instituted. It is shown that this does not hold true, and the explanation is that the faradic stimulus is of too short duration to excite contractions of the musculature, the chronaxia of which has become extended.

My patients have been treated surgically on the basis of the following three indications:

1. *A decompression should be done in cases in which signs of beginning mobility have not appeared after an observation period of two months.*

The reason for this indication is, first, that it has appeared that no patient in whom signs of returning mobility could not be observed within two months obtained complete spontaneous recovery, whereas reports are available that the paresis subsided immediately after a decompression; second, that the paresis may be associated with some other underlying disorder which could not be diagnosed before the operation (e. g., neurinoma).

Of 25 patients belonging to this group, 20 were ready for reexamination. In none of the patients could the paresis be noted when the face was at rest, and they could all close the eye completely and smile and laugh, but to a varying degree; only 2 patients had recovered completely. Even if the decompression is performed a few days after the onset of the paralysis, this is by no means a guaranty that the patient will recover complete mobility. A fact that goes to show that in these patients the improvement may have been due to the operation is that the mobility began rather soon after the intervention, but my material does not supply any decisive proof that the same result would not have been obtained by means of further conservative treatment.

Indication 1 consequently ought to be considered as serving temporarily until better means have been obtained for selecting the early cases for operation.

2. *A decompression should be done if the spontaneous recovery of mobility has ceased before complete restitution has been obtained.*

I have operated on 17 patients, and 13 were ready for reexamination. In 9 the operation had brought about an improvement of mobility, and 1 of these patients had recovered complete mimicry. One of the patients had suffered from a distinct contracture, which disappeared immediately after the operation, and two and one-half years afterward there had been no relapse. At a later date I made the same observation in 2 more cases. This result shows that the contracture is reflex released and is due to an irritation of the facial nerve in the fallopian canal. In 3 patients the decompression was of no effect, but these patients did not get worse. It must be supposed that irreparable injury had already been incurred in the central and peripheral parts of the nerve in patients who did not improve after the operation.

3. *Decompression is indicated in relapsing paresis.*

This group of patients comprises 8 who had had facial palsy from two to nine times, and not one of them had a relapse after the operation. The mobility improved in all of them, and 2 patients recovered completely; none of them had had any tightening sensation in the face after the operation, and all of them had been relieved of enormous psychic pressure.

It seems to be probable that the favorable effect of the decompression is due to the fact that on exposure of the nerve the circulation of the blood is in some way improved by release of the pressure on the vasa nervorum proper or perhaps by the establishment of a collateral circulation.

OSTEOMYELITIS OF THE BASISPHENOID AND BASIOCCIPUT WITH MENINGITIS AND CRANIAL NERVE PALSIES AS A COMPLICATION OF NASOPHARYNGEAL PACKING FOR CONTROL OF EPISTAXIS

Report of a Case with Recovery

ARTHUR A. SPAR, M.D.
Fellow in Otolaryngology and Rhinology, Mayo Foundation
AND
HENRY L. WILLIAMS, M.D.
ROCHESTER, MINN.

OUR PURPOSE in reporting this case is to call attention to the serious complications which may follow the use of nasopharyngeal packing for control of epistaxis.[1] In this instance an abscess of the nasopharyngeal vault occurred and led to osteomyelitis of the basisphenoid and basiocciput, meningitis and cranial nerve palsies. We believe this is the first such case reported in which recovery occurred.

REPORT OF CASE

A 48 year old man came under our care on Dec. 9, 1945, complaining of constant headache, acute pain on swallowing and inability to speak clearly. These conditions had been present for nine weeks. He gave a history of having had severe spontaneous epistaxis nine weeks before admission, which had required continuous anterior and posterior nasal packing for two weeks. Several days after the initial posterior pack was inserted the patient began to have constant headache localized to the vertex and occiput. The headache was of such severity that the administration of merperdine hydrochloride ("demerol hydrochloride") every three hours had failed to give adequate relief. Pain on swallowing and progressive rhinolalia clausa occurred at this time and continued to grow more severe even after all nasal packing had been removed.

On initial examination the patient was apparently in severe pain with evidence of marked loss of weight. He had lost 50 pounds (22.7 Kg.) since onset of the illness. The interior of the nose was normal except for a small area of granulation tissue in the floor of the right nasal passage, which may have been the source of the epistaxis. The nasopharynx was completely occluded by inflammatory edema of the membrane of the soft palate and posterior pharyngeal wall. Creamy pus was running down from the nasopharynx. The tongue deviated to

From the Section on Otolaryngology and Rhinology, Mayo Clinic (Dr. Williams).

1. Spar, A. A., and Hallberg, O. E.: Severe Epistaxis and Its Management: Report of Eleven Cases in Which the External Carotid Artery Was Ligated, Ann. Otol., Rhin. & Laryng. **56**:141-151 (March) 1947.

the right on protrusion. No detectable lymphadenopathy and no meningeal symptoms were present. The temperature was 99.2 F., and it did not rise more than 2 degrees above normal throughout the period of hospitalization.

Complete studies of the blood on admission of the patient revealed apparent pseudohemophilia, which may have been secondary to his current illness, for when later studies were made the hematic values were normal. Roentgenograms of the head, the sinuses, the base of the cranium, the cervical vertebrae and the thorax did not reveal abnormalities at any time.

The patient's condition rapidly became worse. Within twenty-four hours, moderate nuchal rigidity and complete inability to swallow developed, and headache grew more severe.

Neurologic examination revealed right deviation of the tongue, inability to protrude the tongue past the teeth, and weakness or paralysis of the superior constrictor muscle of the pharynx, the levator muscle of the palate and the left masseter and pterygoid muscles. The neurologists concluded that the ninth, tenth and twelfth cranial nerves were involved bilaterally and that the motor part of the fifth cranial nerve was involved on the left side. It was found when spinal puncture was performed that reactive meningitis was present. The concentration of protein in the spinal fluid was 60 mg. per hundred cubic centimeters. Lymphocytes numbered 837 per cubic millimeter and polymorphonuclear leukocytes 400. Sugar and chlorides amounted to 63 and 676 mg., respectively, per hundred cubic centimeters. Initial pressure was 160 mm. of water; final pressure was 120 mm. A differential cell count made after spinal puncture several days later showed that 73 lymphocytes and 44 polymorphonuclear leukocytes were present per cubic millimeter of spinal fluid. The spinal fluid was clear, and neither specimen showed any microscopic evidence of blood. Cultures of the spinal fluid were negative, and a guinea pig inoculation test showed no evidence of the presence of tubercle bacilli. Examination of the fundus oculi revealed no abnormalities.

When specimens of pus obtained from the nasopharynx were cultured, no organisms grew, but we believe that if specimens could have been obtained from within the abscess anaerobic streptococci would have been found. Williams and Heilman [2] in 1937 showed that severe osteomyelitis of the cranial bones is due in most instances to short chain microaerophilic or anaerobic streptococci.

In view of the fact that a pure infection is of such rare occurrence in this area, we felt justified in suspecting that this was a malignant lesion of the nasopharynx with superimposed infection. Both penicillin and sulfadiazine were administered, since in cases of meningitis penicillin may fail to penetrate the hematoencephalic barrier [3] in sufficient concentration to be effective. Penicillin

2. Williams, H. L., and Heilman, F. R.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus, with a Preliminary Report as to Bacteriology and Specific Treatment, Arch Otolaryng. **25**:196-207 (Feb.) 1937.

3. Williams, H. L.: Chemotherapy in Otolaryngology, Wisconsin M. J. **44**: 507-511 (May) 1945. Herrell, W. E.; Nichols, D. R., and Heilman, D. H.: Penicillin: Its Usefulness, Limitations, Diffusion and Detection, with Analysis of One Hundred and Fifty Cases in Which It Was Employed, J A. M. A. **125**:1003-1010 (Aug. 12) 1944. Stern, L., and Rapoport, J. L.: Les rapports entre l'augumentation de la perméabilité de la barrière hémato-encéphalique et les altérations de son substratum morphologique, Compt. rend. Soc. de biol. **98**: 1515-1517, 1928.

was given continuously by the intravenous route, 100,000 Oxford units in each liter of isotonic solution of sodium chloride, and the rate of flow was adjusted so that 200,000 units was given in each twenty-four hour period. Sulfadiazine was given every four hours in doses of 22½ grains (1.5 Gm.), and 60 grains (4 Gm.) of sodium bicarbonate was given with each dose of sulfadiazine. At the time of the second spinal puncture, 50,000 units of penicillin in 20 cc. of isotonic solution of sodium chloride was injected intrathecally.

Radium rays were directed at the nasopharynx and the sphenoid sinuses, 4,000 milligram hours on each of three successive days. It was felt that even if a malignant lesion was not present and the condition was a chronic granuloma, treatment with radium would serve to disrupt the protective barrier around the diseased region and allow the blood-borne antibiotic agents better access to the organisms producing the disorder. It seems probable that our expectations were justified in this instance, because it was not until after the institution of treatment with radium that any improvement could be observed in the patient's condition. Supportive treatment included a transfusion of 500 cc. of blood, feeding by tube and administration of narcotics.

Gradual improvement in all symptoms ensued for several weeks, and nasopharyngeal edema gradually diminished so that it was possible to examine all parts of the vault. A fistulous tract, exuding a steady stream of pus, was seen at the base of the vomer on the upper medial margin of the right choana.

After a ten day period in which the patient's condition appeared to be stationary, further examination was performed and tissue was taken for biopsy while the patient was under anesthesia. Under direct vision with a Yankauer speculum a hemostat was inserted into the fistulous tract and then opened to widen the fistula. A gush of liquid pus followed. A midline incision was made across the vault and downward along the posterior pharyngeal wall, widely exposing the infected region. Grasping forceps were inserted into the lesion in order to remove tissue for biopsy. A mass was encountered which on removal was seen to be a mass of inspissated pus, approximately 2 fluidounces (60 cc.) in volume. The resulting cavity was probed with a finger and found to extend posteriorly throughout the basisphenoid and basiocciput, and bare roughened bone was encountered in all directions. A petrolatum gauze pack was inserted into the cavity for hemostasis. The tissue removed for biopsy was reported by the pathologist as being inflammatory.

The patient improved rapidly thereafter. The nasopharynx healed completely. All symptoms had disappeared except for slight right deviation of the tongue, and the patient was dismissed from the hospital on the fifty-fifth day after admission.

COMMENT

The findings at operation and the clinical picture presented by the patient suggested two papers by Eagleton[4] on osteomyelitis of the sphenoid bone. Although in these papers it was indicated that sphenoid sinusitis was almost always the primary source of infection, the principles applying to a sinal infection that spreads into the basisphenoid

4. Eagleton, W. P.: Suppurative Meningitis of Otitic and Nasal Origin: Its Reaction to Blood Stream Invasion of Pial Vessels, Arch. Otolaryng. **15**: 885-905 (June) 1932; Meningitis from the Sphenoid, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:51-62, 1932.

are equally applicable to infection originating on any surface of the body of the sphenoid bone. In our case the infection originated in the nasopharynx, following trauma and stasis caused by prolonged nasopharyngeal packing. The basisphenoid depends on the adjacent periosteum for its blood supply; therefore any embarrassment of its blood supply may leave it vulnerable to necrosis and infection. Trauma and infection of the nasopharyngeal mucosa probably led to thrombophlebitis of the vessels supplying this bone, and osteomyelitis resulted.

The preexistence of an infection of the pharyngeal bursa or of the craniopharyngeal canal could not be determined. A brief review of the literature on these entities [5] showed no report of a lesion such as occurred in our case. Druss,[6] in 1929, reported a case of meningitis in which the patient died as a result of disease of the craniopharyngeal canal, but the body of the sphenoid bone was not destroyed grossly. Many others [7] have reported cases of osteomyelitis of the sphenoid bone secondary to sphenoid sinusitis or apicitis, in which almost all the patients died.

We consider the superior margin of the right choana, well anterior to the adenoid region and inferior to the ostium of the sphenoid sinus, to have been the site of origin of the abscess. The size of the cavity in the basisphenoid was evidence that the overlying meninges must have been in close contact with the inflammatory process. The products of inflammation extending into the meninges in this region gave rise to the reactive meningitis. The possibility that there was a nonunited metaphysis in the basisphenoid as described by Harris [8] suggests an additional factor in the development of the meningitis. That portion

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5. Yankauer, S.: Nasopharyngeal Abscess: Report of One Hundred and Fifty-Five Cases, Tr. Am. Acad. Ophth. **34**:364-375, 1929. Hollender, A. R., and Szanto, P. B.: Thornwaldt's Syndrome, Ann. Otol., Rhin. & Laryng. **54**: 575-581 (Sept.) 1945. Kully, B. M.: Cysts and Retention Abscesses of the Nasopharynx: A Report of Eighty-Eight Cases, Tr. Am. Laryng., Rhin. & Otol. Soc. **40**:185-199, 1934.

6. Druss, J. G.: Intracranial Complications Associated with Disease of the Sphenoid Bone: Report of Two Cases: (1) Meningitis Secondary to Infection of the Craniopharyngeal Canal (?) and (2) Abscess of the Frontal Lobe, Arch. Otolaryng. **10**:39-48 (July) 1929.

7. Ballenger, H. C.: Osteomyelitis of the Sphenoid Bone: Report of Five Cases with the Autopsy Findings, Ann. Otol., Rhin. & Laryng. **48**:95-102 (March) 1939. Brunner, H.: Chronic Osteomyelitis of the Skull, ibid. **52**:850-894 (Dec.) 1943. Lawson, L. J.: Osteomyelitis of the Sphenoid Bone: A Report of Two Cases, Arch. Otolaryng. **25**:1-10 (Jan.) 1937. Kaufman, A., and Hartmere, S. J.: Latent Osteomyelitis of the Sphenoid Bone Reactivated by Trauma, with Death from Meningitis, Laryngoscope **44**:477-487 (June) 1934.

8. Harris, H. A.: The Closure of Cranial Sutures in Relation to the Evolution of the Cortex Cerebri and Aphasia, Proc. Staff Meet., Mayo Clin. **2**:206-208 (Aug. 31) 1927.

of the medulla oblongata from which the ninth to twelfth cranial nerves originate lies on the basiocciput. Involvement of the medulla can be assumed to have given rise to the neurologic symptoms. The involvement of the masseter and pterygoid muscles was transient and was probably the result of limitation of motion due to pain.

The diagnosis was not apparent immediately. The absence of roentgenologic changes suggested that this was basically an inflammatory process. Simple acute nasopharyngitis was ruled out at once by the duration and the progressive nature of the disease. Tuberculoma and tuberculous meningitis were excluded by the bacteriologic study, the examination of the spinal fluid and the roentgenogram of the thorax. Intracranial hemorrhage was excluded by the examination of the spinal fluid and by the febrile course of the disease. A malignant lesion of the nasopharynx or of the sphenoid sinus with superimposed infection seemed a possible diagnosis in the critical period of the patient's illness; so radium was used. Even though the lesion later proved to be nonmalignant, the benefit of the use of radium in facilitating drainage became evident when the amount of pus coming from the nasopharynx increased noticeably within twenty-four hours after the use of radium. This observation concurs with the impressions published in 1942 by Williams and Popp.[9]

9. Williams, H. L., and Popp, W. C.: Roentgen Therapy for Acute Sinusitis, Radiology **38**:290-293 (March) 1942.

SYMPOSIUM ON FENESTRATION OF THE LABYRINTH

LEMPERT FENESTRA NOV-OVALIS OPERATION FOR THE RESTORATION OF SERVICEABLE UNAIDED HEARING IN PATIENTS WITH CLINICAL OTOSCLEROSIS

Its Present Evolutionary Status

JULIUS LEMPERT, M.D.
NEW YORK

THAT great progress has been made toward the ultimate solution of the surgical problem of improving hearing in patients with clinical otosclerosis is borne out by the fact that every interested otologist knows today: first, that hearing can be improved to the practical, serviceable level for both social and economic use in a good percentage of deafened persons by performing the fenestra nov-ovalis operation for clinical otosclerosis and, second, that many such newly created vestibular fenestras can and do stay permanently open, with the result that the hearing is permanently maintained at this high level.

The ten year postoperative audiogram of M. E. (fig. 1) demonstrates the fact that a newly created vestibular fenestra can remain patent and that practical, serviceable hearing when obtained by fenestration can be continuously maintained at that high level.

Damage of the organ of corti secondary to postoperative labyrinthitis is no longer the threat it formerly was. By creating and ultimately sealing the fenestra in a blood-free surgical field and by using prophylactically a single daily dose of 300,000 units of penicillin for a period of ten days its incidence has been reduced to 1 per cent or less. However, the two inevitable questions concerning this surgical procedure which have been waiting to be answered to the complete satisfaction of the patient are:

1. Will his personal hearing acuity be improved to the level necessary to insure practical, serviceable unaided hearing for both social and economic contacts?

2. If his hearing is improved to the practical, serviceable unaided level, will it remain at that high level permanently?

In other words, he exhibits little interest in receiving as a reward for subjecting himself to this operation an improvement of the audiometric reading of 10 decibels or more if the reading is not representative of the result he asked for and hoped for, namely, practical, serviceable

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unaided hearing permanently maintained. Though he is fully aware of the fact that the over-all percentage of permanent practical, serviceable end results obtainable with this operation is today quite high, he fails to enthuse over this fact if he happens not to be one of the lucky beneficiaries thereof. He has, however, always been willing to submit to this operation in its individually unpredictable state with full understanding of the existing uncertainties as to his personal end result, because he knows that this operation is the only one available for having his hearing permanently improved to the practical level and that the odds of obtaining such a result, in spite of the individually unpredictable state of the operation, are nevertheless considerably in his favor.

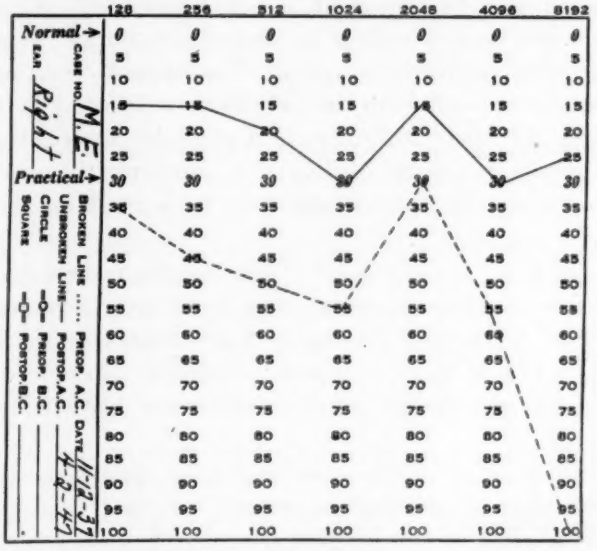

Fig. 1.—Audiogram of M. E. It demonstrates that a newly created vesticular fenestra can remain open and that the practical, serviceable hearing obtained by fenestration can be continuously maintained at that level over a period of ten years.

Why have the two most vital questions advanced by the patient contemplating his submission to the fenestration operation not been answered to his satisfaction?

The reasons why the individual end result of the fenestration operation remains in the nonpredictable state are twofold:

1. Although with the tests available at present one can in the majority of cases determine preoperatively the likelihood of restoring practical, serviceable hearing as a result of fenestration, these tests, being all subjective, are not always infallible.

2. Although, owing to the advances made in this form of surgical treatment for clinical otosclerosis in the last ten years, the newly created

fenestras have remained permanently open in a large percentage of cases, otologists have, nevertheless, had no way of knowing in advance which ones will remain patent.

What has been done to accomplish the advance of the surgical treatment for clinical otosclerosis from the originally pioneering, unfavorable status to its present over-all highly favorable status?

In 1938[1] I first described and advocated the use of a one stage technic for fenestrating the external semicircular canal posterior to its ampulla which when employed in a suitable case of clinical otosclerosis could result in an improvement of air conduction such that the threshold of hearing would be raised to the 30 decibel or higher level under normal in the three most important speech frequencies—512, 1024 and 2048 cycles per second—which is tantamount to the restoration of practical, serviceable unaided hearing. Simultaneously with my description of this technic I set forth the indications which may be considered as suggesting that there still exists in a given ear deafened as a result of otosclerosis the potential possibility for improving the air conduction hearing to the practical, serviceable level as a result of this surgical treatment.

Under the title "Indications" I stated that the technic described for the improvement of hearing might be employed with good results when there existed ". . . normal hearing by bone conduction, determined by masking, or a loss in hearing by bone conduction, determined by masking, which does not exceed 30 decibels for the 512, 1024 and 2048 frequencies."

The importance of strictly observing these indications as set forth if the restoration of practical, serviceable unaided hearing is to be expected as a result of this surgical treatment has since been corroborated by all otologists doing this operation.

When otologists confine their surgical efforts for clinical otosclerosis to those cases which meet the aforementioned suitability requirements, the chances of obtaining practical, serviceable unaided hearing as a result of surgical fenestration are greatly enhanced.

In my attempts to increase the over-all percentage of postoperative permanent practical and serviceable end results, I advocated and employed means and ways of eliminating from the technic everything that could aid and abet the naturally existing tendency toward osteogenetic repair and closure of the newly created fenestra, which prevents the permanent maintenance of the originally obtained improvement of hearing.

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1. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis: A New One Stage Surgical Technic, Arch. Otolaryng. **28**:42-97 (July) 1938.

I advocated first that this surgical procedure be performed with the least amount of traumatization of the tissues involved in order to reduce the postoperative inflammatory reaction to the minimum. Severe postoperative inflammation encourages rapid osteogenetic repair of the freshly created fenestra.

In the April 1941 [2] issue of the *Laryngoscope* I stated:

> Postoperative inflammatory reaction of the soft tissues is the greatest deterrent to the permanent restoration of serviceable hearing in otosclerosis. It is for this reason that every step in the technique must be performed with the least amount of tissue sacrifice and the least amount of trauma to the tissues involved. This surgery must be performed under the strictest rules of asepsis and the postoperative wound must always be treated with sterile precautions. Unless the postoperative inflammatory reaction of the tissues is held down to an absolute minimum, new bone regeneration will take place and close the external semicircular canal fenestra in spite of all the technical precautions taken to prevent osteogenesis. The most serious sequel of excessive postoperative inflammatory soft tissue reaction is serous labyrinthitis.

Second, believing that when a freshly injured bony surface is covered with a periosteum-lined membrane this will aid and abet formation of new bone, I attempted to cover the newly created fenestra with the epithelium-lined surface of Shrapnell's membrane.

Third, bone sand and bone slivers, both by-products of this operation which when left behind can accelerate bone regeneration, were always carefully removed from the area of the fenestra and the rest of the surgical field.

I quote from the ARCHIVES OF OTOLARYNGOLOGY of May 1940 [3]:

> All particles of bone dust must be removed from the floor and the lateral bony walls of the newly created fenestra.

However, every otologist who has done the fenestration operation in more than one human ear and every investigator who has done animal research pertaining to the subject and has done this research with controls knows today that neither the secondary removal of bone dust nor the primary, initial absence of bone dust, which needs no removal, can prevent osteogenesis from taking place in the freshly cut bony walls forming the rim of the fenestra.

Fourth, I attempted to discourage the formation of a connective tissue matrix within the gap of the newly created fenestra by which osteogenesis could rapidly spread from the freshly cut bony fenestral

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2. Lempert, J.: Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis: Indications, Technique, Observations as to Early and Late Postoperative Results, Laryngoscope **51**:330-362 (April) 1941.

3. Lempert, J.: Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis: Summary Report of One Hundred and Twenty Cases, Arch. Otolaryng. **31**:711-779 (May) 1940.

rim in its mission of repair of the fenestra. This was accomplished (*a*) by controlling bleeding in the surgical wound and creating the fenestra in an absolutely bloodless field; (*b*) by never sealing the fenestra with the tympanomeatal membrane in the presence of even the most infinitesimal bleeding anywhere in the surgical wound; (*c*) by changing the site of the fenestra. As a result of observations made during an extensive study and investigation of the part played by the fibrous connective tissue matrix within the fenestral gap in aiding and abetting osteogenesis in its mission of repair and closure of the fenestra, I developed, described and since 1941 have exclusively employed the fenestra nov-ovalis technic [4] for fenestrating directly into the vestibule instead of into the lumen of the external semicircular canal posterior to the ampulla, hoping thus to prevent the formation of a fibrous connective tissue matrix within the fenestral gap.

By removing the incus plus the head and neck of the malleus and fenestrating the vestibule posterolateral to the oval window niche, with only the fallopian canal separating the fenestra nov-ovalis from nature's oval window, I created a fenestra communicating directly with the wide and deep perilymphatic space of the vestibule, which is 3 mm. wide and 5 mm. deep. A perilymphatic cisterna of such dimensions makes it much more difficult for a fibrous connective tissue matrix to form and bridge the fenestral gap than it was when the lumen of the external semicircular canal was fenestrated posterior to the ampulla and the newly created fenestra communicated with the narrow and shallow perilymphatic space, the circumference of which is only about 0.8 mm.

The fenestra nov-ovalis technic has made it possible to improve air conduction hearing to the practical and serviceable level necessary for both social and economic contacts in about 80 per cent of the most carefully chosen suitable cases of clinical otosclerosis. However, the continuous maintenance of practical and serviceable unaided hearing for more than two years, and permanently thereafter, has been obtainable in only about 75 per cent of such postoperatively successfully improved ears.

Although there are reports in the literature that much higher percentages of permanently maintained improvements of 10 decibels or more have been obtained with the fenestra nov-ovalis operation, it should be understood that in a large proportion of the patients, though the audiometric reading was improved 10 decibels or more, the hearing

4. Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880-912 (Nov.) 1941.

improvement has not reached the level necessary to make it either practical or useful for social and economic contacts.

The degree and the extent of the air conduction loss, no matter how great it may preoperatively be, will nevertheless not interfere with the possibility of restoring the hearing to the practical and serviceable unaided level with the use of the fenestra nov-ovalis operation provided the masked bone conduction hearing for the 512, 1024 and 2048 frequencies has remained unimpaired.

Normal hearing can sometimes also be restored in the patient with clinical otosclerosis by vestibular fenestration and the hearing continuously kept at this level. This can be accomplished even in an ear

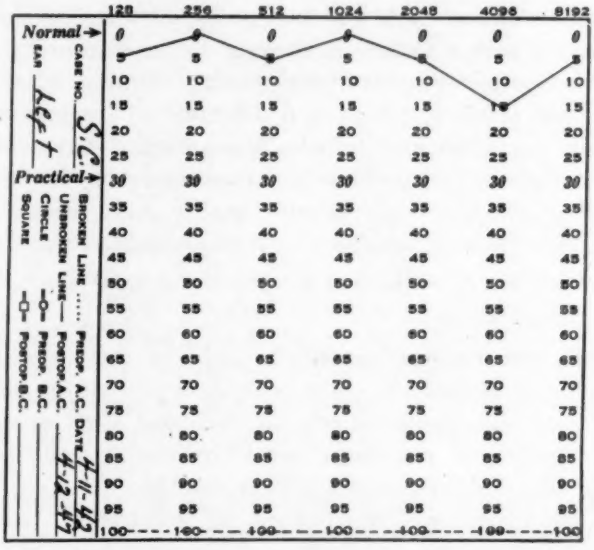

Fig. 2.—Audiogram of S. C. It shows that hearing restored to normal in an ear that preoperatively was totally deafened as regards air conduction was maintained at that high level five years after fenestration.

which preoperatively is totally deafened as regards air conduction if the cochlear nerve function has remained normal. The five year postoperative audiogram of S. C. (fig. 2) is offered in proof of this.

It is today generally agreed by all otologists doing fenestrations that when strictly adhering in their choice of cases to the indications as set down and employing the fenestra nov-ovalis technic, they have obtained hearing improvement that reached the practical, serviceable level of 30 decibels and higher and remained at these levels for two years, and continuously thereafter, in a much larger percentage of cases than ever before. If, however, the surgical treatment for the restoration of practical and serviceable unaided hearing is to serve both beneficially

and permanently a still greater percentage of those deafened as a result of otosclerosis, then there will have to be developed:

1. A practical and specific objective test for the accurate preoperative determination of the unused reservoir of cochlear nerve function existing in the ear chosen for fenestration. With such objective testing one could prognosticate preoperatively whether the hearing in an individual ear will be improved to the practical level in consequence of fenestration.

2. A technic embracing a nondisputable, definitely proved specific means, arrived at as a result of scientifically controlled experimental animal research, for deliberately preventing osteogenesis from arising postoperatively in any of the three histologic layers of the freshly injured bony walls of each and every newly created fenestra. The development of such a technic is essential because, in order to be able to assure the patient preoperatively that the hearing when improved to the practical, serviceable level by fenestration will remain at that level permanently, one must first technically assure the permanent patency of every one of the labyrinthine fenestras created.

Only thus can the nonpredictable quality and permanency of an individual end result be advanced to the reasonably predictable status.

What has been accomplished toward the development of objective means of testing hearing?

Although with the hearing tests available at present and the commonly employed technics of these tests it is possible in the majority of cases to determine preoperatively the likelihood of restoring practical, serviceable hearing in a deafened ear by vestibular fenestration, these tests, being all subjective, are not always infallible.

Since the advent and employment of fenestration, with the end result dependent on the preoperative determination of the suitability of the ear in question, it became essential and imperative that more accurate, more dependable means of determining the precise degree and type of hearing loss existing preoperatively in a given ear be devised.

In testing the hearing of deafened persons seeking relief by vestibular fenestration the important thing is to determine not how long they say they hear a particular tuning fork by air or by bone conduction but rather to determine accurately the moment when they actually do cease to hear the tuning fork. The determination of auditory acuity cannot be left entirely to the subjective response of the patient since this often leads to an erroneous estimation of the hearing status.

Cognizant of the fact that the future economic and social welfare of the patient would depend on my diagnosis and advice, I was led by my anxiety to determine the exact degree of cochlear nerve function

to the observation and conclusion that the subjective responses of many patients to tuning fork tests, for reasons not unexplainable, are often unreliable. Convinced as I am that to date otologists still have no better method of determining the degree of unused function of the cochlear nerve in an air conduction–deafened ear than the careful and intelligent use of the 512, 1024 and 2048 steel tuning forks, I have for many years been searching for means of interpreting the results of such testing objectively without depending on the voluntary responses of the patient.

An Objective Method of Testing Hearing with Tuning Forks: For reasons best known to themselves, many patients seeking to have their hearing improved with the fenestration operation believe, or wish to make the examiner believe, when they are being tested for suitability, that they hear the various tuning forks by bone conduction much longer than they actually do, and will indicate by hand or word of mouth that they no longer hear the tuning fork long after they actually ceased hearing it.

By insisting that when being tested the patient must not bend his head or shut his eyes and must face the examiner with chin up and eyes widely open, I was able to observe that simultaneously and synchronously with the cessation of hearing the eyes deviate involuntarily to the side and in the direction of the tuning fork. In very many patients this involuntary deviation of the eyes takes place much sooner than their decision to indicate by hand or word of mouth that they no longer hear. This involuntary deviation of the eyes, I concluded, was due to the patient's search for the sound which he had just lost. This conclusion was readily corroborated and confirmed on questioning of thousands of patients.

With acceptance of the earlier appearing objective involuntary deviation of the eyes as the hearing limit for a particular tuning fork instead of the often voluntarily delayed and belated subjective response of hand or mouth, a much more accurate determination of the existing degree of cochlear nerve function is possible. With employment of this objective test it is possible to predict preoperatively with much greater accuracy the possibility of restoring practical hearing in the patient with clinical otosclerosis.

What has been done toward the development of a specific means for the prevention of osteogenetic closure of the newly created fenestra?

In order to advance the fenestra nov-ovalis from the individually nonpredictable to the individually reasonably predictable status, I attempted to prevent osteogenesis, the major factor of defeat, from taking place locally in the freshly injured bony walls of each and every one of the labyrinthine fenestras created, so that the permanency of each indi-

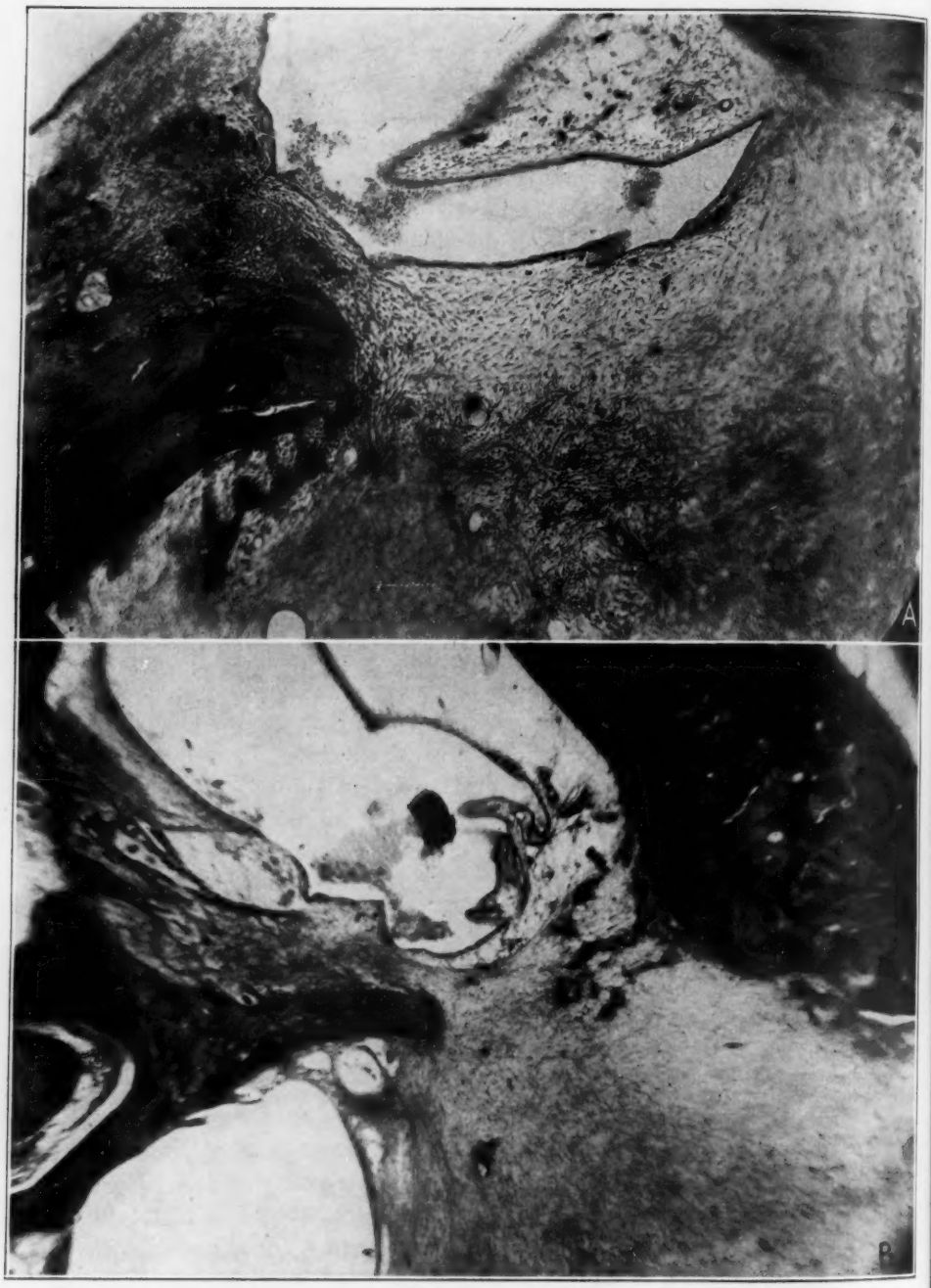

Fig. 3.—*A*, periosteal bone advancing toward the rim of the fenestra twelve days after operation (left ear of monkey 13).

B, endosteal bone appearing eighteen days after operation (left ear of monkey 16, experiment I).

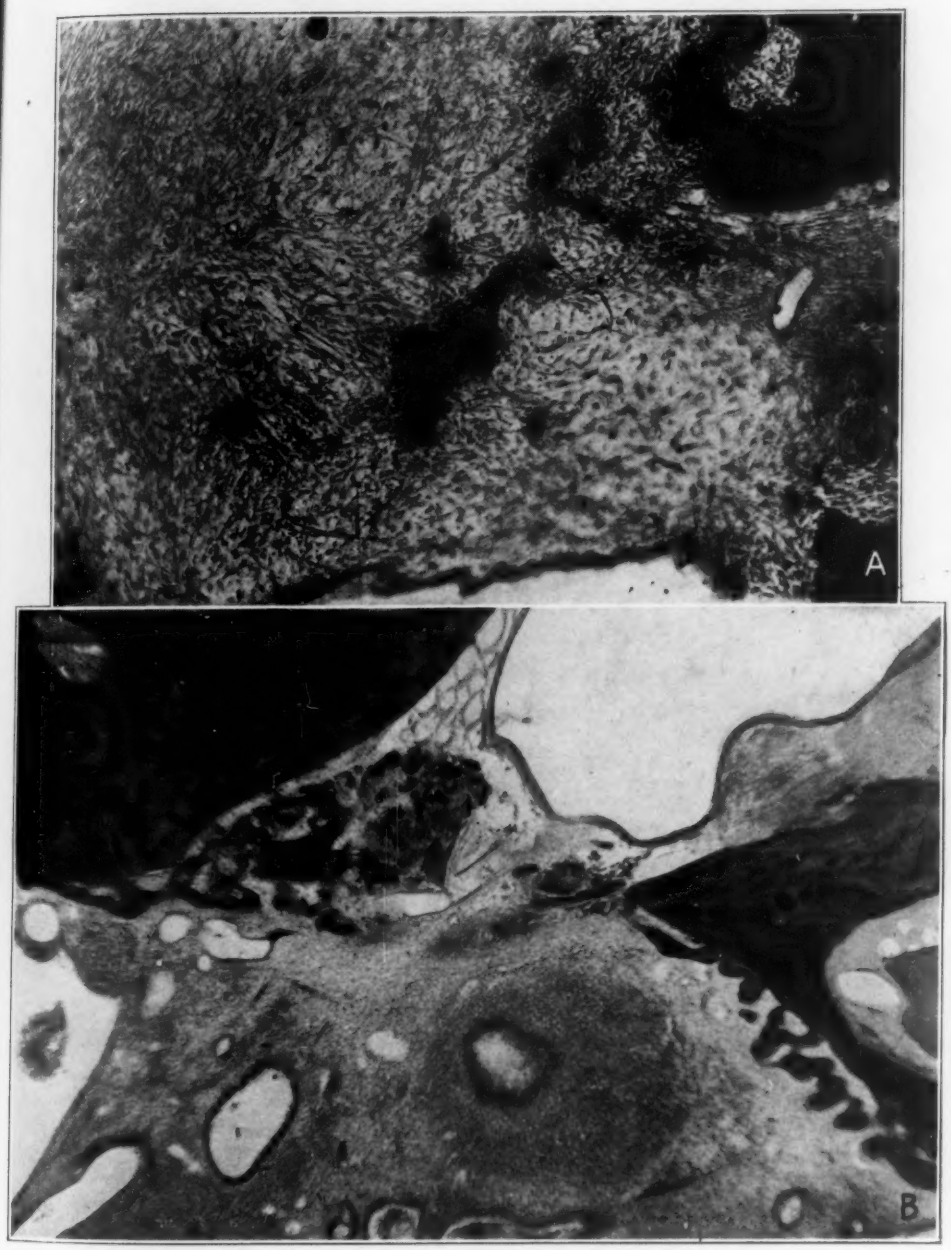

Fig. 4.—*A*, islands of new bone appearing in the fenestrated area fourteen days after operation (left ear of monkey 15).

B, enchondral bone regenerating across the cut edges of the otic capsule twenty-two days after operation (right ear of monkey 17 B (92), experiment I).

Fig. 5.—*A*, enchondral bone, as well as endosteal and periosteal bone, participating in regeneration (right ear of monkey 19, experiment II, with polishing burr). *B*, high power magnification of regenerating enchondral bone.

vidual improvement of hearing obtained by fenestration could be forecast and reasonably assured.

Gross observations made in human ears during revisions of osteogenetically closed newly created labyrinthine fenestras and histologic studies of bone regenerating within newly created labyrinthine fenestras in rhesus monkeys have revealed that osteogenesis takes place in the endosteal, periosteal and enchondral layers of the fenestral rim. The repair of the endosteal layer is, as a rule, the first and fastest, while that of the enchondral layer is usually the slowest. Given time, regeneration ultimately involves all three layers (figs. 3 to 5).

Fig. 6.—Fenestra closed by endosteal bone forming beneath the tympanomeatal flap four months ten days after operation (monkey 16, experiment III).

In view of the aforementioned observations made in both human and monkey ears it became quite obvious that if a newly created fenestra was to remain permanently patent, means and ways of locally hindering osteogenetic repair within all the three histologic layers of the bony rim of the fenestra, or whatever layers were left after creating the fenestra, would have to be devised. It is erroneous to reason and suggest that if the outer, periosteal layer were extensively removed beyond the margins of the fenestral gap down to the enchondral layer this removal would in any way prevent osteogenetic closure of the newly created fenestra. Just as long as the inner, endosteal layer, which cannot be removed

beyond the margins of the fenestral gap, forms part of the fenestral rim, osteogenesis will take place within the endosteal layer and close the gap. Once the endosteal layer regenerates it is inconsequential whether the enchondral layer remains patent or whether the periosteal layer has been extensively removed. Osteogenetic repair of any one of the three histologic layers of the fenestral rim, with the other two layers not participating in the process of repair, will naturally result in closure of the fenestra and defeat of the objective of permanently maintaining the

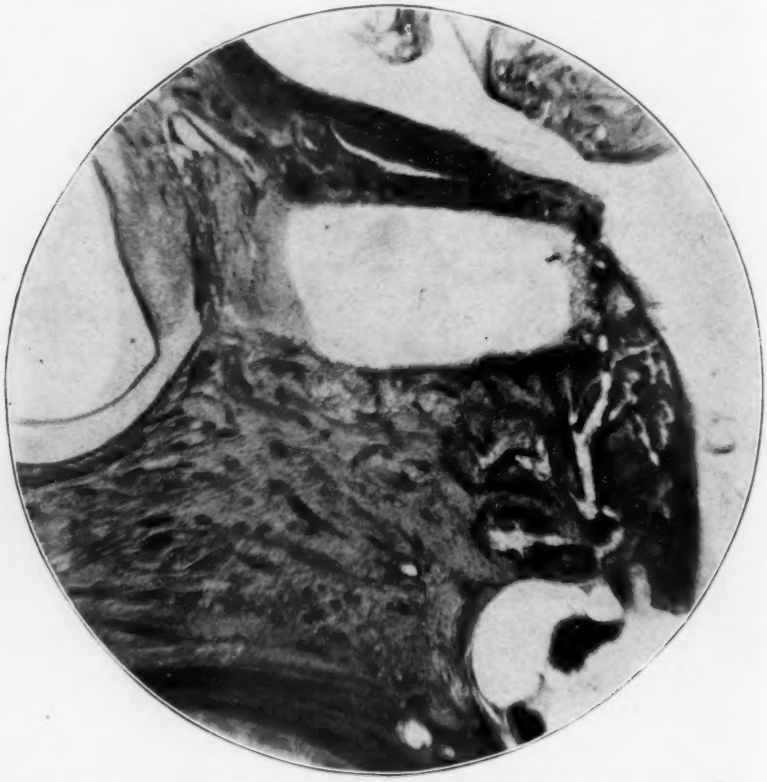

Fig. 7.—The endosteum has been completely removed, and there is regeneration of periosteal bone only, six months one week after operation.

hearing improvement following fenestration, just as much as if all the layers participated and regenerated (fig. 6).

From the observations made in human ears and the experimental histologic studies of rhesus monkeys it became evident that neither removing the endosteum from the region of the newly created fenestra nor leaving it intact prevents osteogenetic closure of the fenestra.

The belief that disturbance or removal of the endosteum facing the perilymphatic space beyond the fenestral region would necessarily result

in formation of new bone was not substantiated by animal experimentation. Histologic studies of rhesus monkeys showed that injury of the endosteal lining of the perilymphatic space does not necessarily result in bone regeneration and that the avoidance of injury of the endosteum does not necessarily prevent bone regeneration in the perilymphatic space (figs. 7 to 10).

Believing as I always did that a permanently patent fenestra could be deliberately created by locally preventing the start of osteogenesis

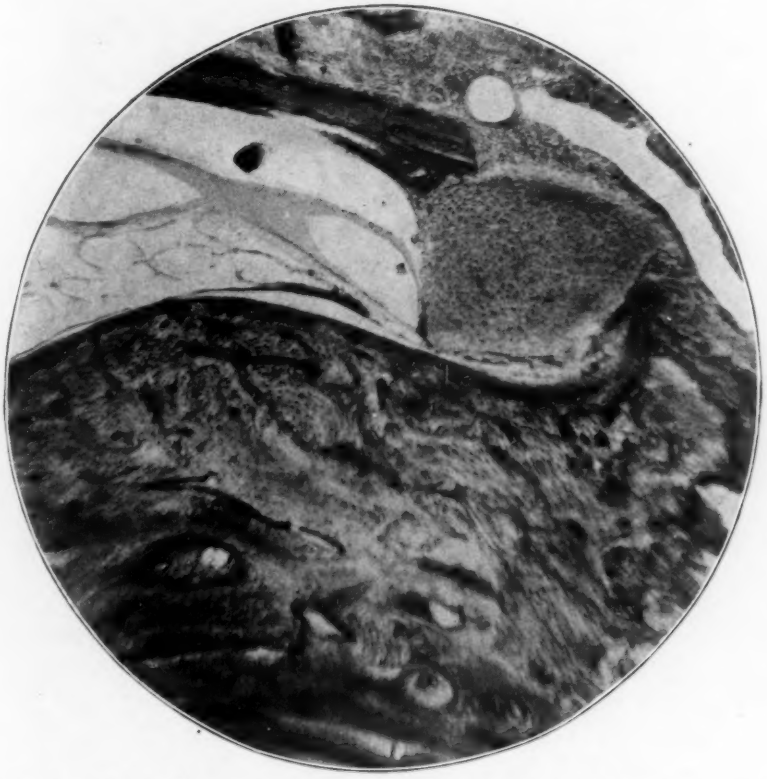

Fig. 8.—The endosteum was torn from the bony wall. There is no regeneration at this site four months ten days after operation.

in the freshly injured bony walls of the fenestra and that this must be accomplished if the fenestra nov-ovalis operation was to be brought to its final successful conclusion, I attempted to reach this objective by basing my research in both human and animal subjects on the following two surgical principles:

1. Physical impedance must be offered to the osteogenesis that starts and spreads from the normal histologic structure of the freshly cut

periosteal, enchondral and endosteal layers of the fenestral rim, and the rim thus forced to heal without osteogenetic repair.

2. The histologic structure responsible for osteogenesis within the freshly cut margins of the periosteal, enchondral and endosteal layers of the bony fenestral rim must be devitalized and inactivated without being destroyed.

In my attempt to force healing of the freshly cut bony walls of the fenestra without osteogenetic repair by instituting physical impedance

Fig. 9.—Regeneration of endosteal bone in the presence of mature fragments or bone dust six months twenty-eight days after operation.

for the prevention of the onset and spread of osteogenesis I employed the following means:

1. Soft tissue impedance: When covering and sealing the newly created fenestra I pressed the tympanomeatal membrane tightly into the fenestral gap in the hope of physically preventing the osteogensis of the bony rim from spreading and subsequently closing the gap.

In the July 1938 issue of the ARCHIVES OF OTOLARYNGOLOGY, under the heading "Insertion of the Tympanomeatal Cutaneous Membrane

into the Troughlike Fenestra of the External Semicircular Canal," I [1] stated:

A piece of paraffin mesh rolled into a small ball is placed over the portion of the tympanomeatal membrane which covers the outer aperture of the troughlike fenestra in the external semicircular canal. After gentle pressing and molding of the paraffin mesh over Shrapnell's membrane, the latter is inserted into the troughlike fenestra so as to line the inner surface of its walls, extend immediately beyond the edge of the inner aperture of the fenestra and lie in direct contact with the perilymph. The paraffin mesh ball is held in place by additional pieces of paraffin

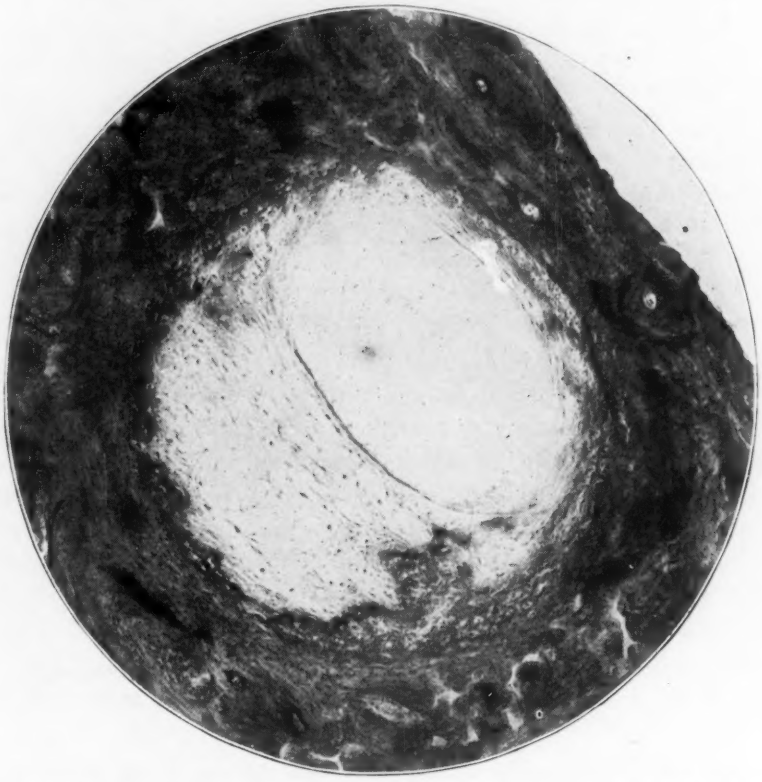

Fig. 10.—Regeneration of endosteal bone, with perilymphatic tissue and endosteum intact and with no mature bone dust or fragments present, four months ten days after operation.

mesh inserted into the mastoid wound. On the care and precision with which this step in the technic is carried out depends the permanent maintenance of the fenestra in the bony capsule. The insertion of Shrapnell's membrane into the troughlike fenestra in the manner described is another step in the mechanical prevention of closure of the fenestra by regeneration of bone.

In the January 1946 issue of the ARCHIVES OF OTOLARYNGOLOGY, under the title "Histologic Observations on the Healing of Labyrinthine

Fistulas in Monkeys," Lindsay [5] showed in his figure 8 a photomicrograph of an experimental fistula which had been present in the right ear of monkey 30 for one hundred and sixty-three days. In his description of this photomicrograph he pointed out that there was no sign of active regeneration of bone at the margins of the fistula and that the covering flap was attached to the membranous canal and separated from the perilymphatic space by it and new bone along the margins of the fistula.

On the basis of this observation made in his animal experiments he concluded as follows:

> The evidence suggests that any fistula remains open only by virtue of the union which occurs between the membranous labyrinth and the covering flap to the exclusion of osteogenetic tissue.

In the November 1941 issue of the ARCHIVES OF OTOLARYNGOLOGY I [4] published a series of photomicrographs of a sectioned temporal bone removed from one of my patients two weeks after the labyrinth had been fenestrated for clinical otosclerosis, which showed complete absence of bone regeneration and the wide patency of the newly created fenestra. I also called attention to the observation made that the endolymphatic labyrinth had become adherent to the tympanomeatal membrane which covered the fenestra (fig. 11).

However, if I were to draw a conclusion from this observation I should take into consideration all the other findings and observations which I have made in the living human subject and in many of the monkeys and in some of the published photomicrographs of sections published by various authors reporting their results with experimental fistulization of the animal labyrinth and be forced to reach a conclusion which is the complete opposite of the one reached by Lindsay. Instead of concluding as he did, I should conclude as follows:

> The evidence suggests that any union which occurs between the membranous labyrinth and the covering flap to the exclusion of osteogenetic tissue occurs only by virtue of the fistula's remaining open.

I question the possibility that adhesion can be deliberately caused between the epithelium-lined endolymphatic labyrinth and the periosteum-lined tympanomeatal membrane without the epithelium-lined outer surface of the endolymphatic labyrinth being traumatized first. I also question the effect that such adhesion would have, when and if brought about in the human subject, on the desired hearing improvement for which this operation is meant. By carrying the investigation of this phase a little further, beyond the observation of an occasional adhesion of membranous labyrinth and tympanomeatal flap, it can be readily

5. Lindsay, J. R.: Histologic Observations on the Healing of Labyrinthine Fistulas in Monkeys, Arch. Otolaryng. **43**:37-48 (Jan.) 1946.

observed that when and if the endolymphatic labyrinth becomes adherent to the tympanomeatal flap it can remain so only just as long as bone regeneration for some unknown reason does not take place. However, if bone regeneration takes place, it will spread knifelike across the fenestral gap and again separate the endolymphatic labyrinth from the flap. Adhesion of membranous labyrinth and tympanomeatal flap is not a means of preventing osteogenetic closure of the newly created fenestra but rather is a result encountered occasionally because osteo-

Fig. 11.—Open fistula in the human ear showing the tympanomeatal flap adherent to the endolymphatic (membranous) labyrinth. The highly polished edges of the bony rim of the fenestra are clearly seen to be involved in the fibrotic process of repair. This condition was present fourteen days after operation.

genesis did not take place for some unknown reason and the fenestra remained either completely or partially patent. In figure 12 a photomicrograph of a histologic section of a fenestrated ear of a rhesus monkey is offered in support of my contention.

Further proof that adhesion of the covering membrane and the endolymphatic labyrinth is not a means of preventing bony closure of the fenestra was furnished by the fact that the highest percentage of osteo-

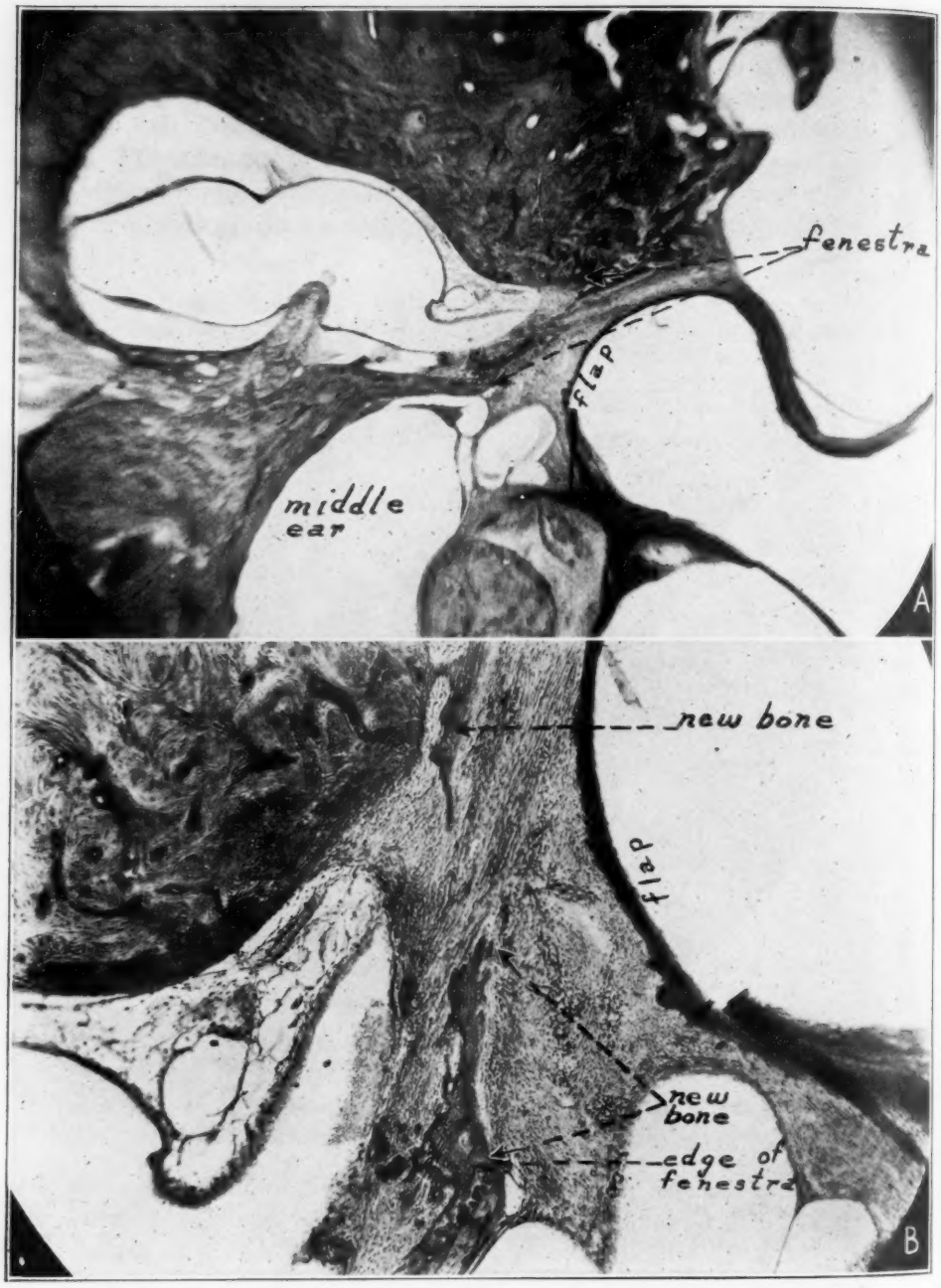

Fig. 12.—*A*, endosteal bone growing, knifelike, across the fenestra and separating the membranous labyrinth from the tympanomeatal flap, to which it had become adherent (monkey 7, experiment III).

B, high power magnification.

genetic closures of fenestras resulted prior to the employment of the fenestra nov-ovalis technic, when the fenestra was made into the shallow and narrow, 0.8.mm., lumen of the external semicircular canal posterior to the ampulla, a site most favorable for inducing adhesion between the endolymphatic labyrinth and the tympanomeatal membrane.

2. Physical impedance with an inert metal obturator frame: In the November 1941 issue of the ARCHIVES OF OTOLARYNGOLOGY I [4] described a technic which I had employed experimentally in a small series of cases, which I called "fenestra nov-ovalis with obturator frame." I stated:

> The comparatively large dimensions of the perilymphatic space in the region of the fenestra nov-ovalis offer an opportunity for inserting and permanently maintaining in direct contact with the lateral bony walls of the fenestra an inert metal obturator frame, in order to mechanically prevent the fenestra from closing.

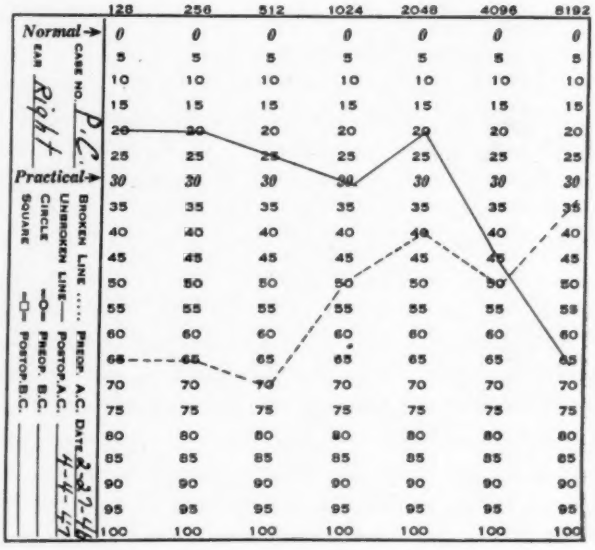

Fig. 13.—Audiogram of P. C. This patient's fenestra was kept open with a metal prosthesis.

There is no doubt in my mind today that an inert metal obturator frame snugly fitted to the bony rim of the newly created fenestra can remain in situ and prevent osteogenesis. However, not until a method for creating a fenestra of standard size and shape in the bony labyrinthine capsule is developed, so that a prefabricated inert metal frame of the same size and shape can always be inserted with ease, will the use of the metal frame obturator become practical for the mechanical and physical prevention of bony closure of the fenestra (fig. 13).

3. Physical impedance with a cartilage stopple: After having demonstrated through extensive research that a cartilage stopple inserted into

the newly created fenestra to separate the postoperatively inflamed tympanomeatal membrane from the endolymphatic labyrinth within the perilymphatic space could prevent postoperative labyrinthitis with damage of the organ of Corti, and that when the cartilage stopple was inserted into the fenestral gap loosely so that it remained mobile it functioned like a normal stapes and permitted the perilymph and the endolymph to be mobilized by air-borne sound, I described the fenestra nov-ovalis with mobile stopple technic in the January 1945 issue of the ARCHIVES OF OTOLARYNGOLOGY.[6]

I employed this technic in 750 cases, and abundant evidence exists today that the mobile cartilage stopple technic when correctly applied prevents severe postoperative inflammation of the endolymphatic labyrinth with damage of the organ of Corti and also prevents osteogenetic closure of the newly created fenestra.

The careful inspection of 48 healed fenestras with mobile cartilage stopples in situ has provided conclusive and nondisputable proof that when the stopple is inserted into the gap of the fenestra so that both its inner surface, facing the perilymphatic space, and its outer surface, facing the epitympanum, would respectively reach and extend beyond the endosteal and periosteal surfaces of the bony rim of the fenestra as found in 46 of the 48 cases cited, osteogenesis can be prevented.

In none of the 46 cases was any evidence of bone regeneration observable, and when the stopple was removed the fenestral rim was always found completely healed and the fenestral gap as widely patent as it was when originally created. The endolymphatic labyrinth appeared healthy and unchanged. The cartilage stopples which had been properly inserted in position and which had remained in position showed histologically no signs of having undergone any changes. Two were not correctly placed, and in these bone regeneration had taken place (fig. 14).

As encouraging as the foregoing picture of my findings appears in itself to be, I am sorry to state that despite the fact that the mobile cartilage stopple technic when correctly employed succeeds in preventing bone regeneration, fenestration surgery was nevertheless not advanced to the individually predictable status as a result of its use because the question which the patient would like to have answered—namely, will his hearing when and if restored to the practical level remain at that level permanently?—still remains unanswerable to the patient's satisfaction.

In a considerable percentage of cases the use of the mobile cartilage stopple has created an entirely new difficulty, heretofore never encountered, which prevents the mobile cartilage stopple technic from becoming the final solution to the problem of surgical fenestration.

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6. Lempert, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. **41**:1-41 (Jan.) 1945.

In about 25 to 30 per cent of the patients successfully operated on with the mobile cartilage stopple technic, the improved hearing began to show signs of recession and gradually returned to the preoperative level within periods varying from four to twelve months after operation. Revisions of the newly created fenestra and inspection with Zeiss magnifying lenses invariably revealed that the cartilage stopple was almost always unchanged and in situ but was no longer as freely mobile as is necessary for its mobility to be utilized by air-borne sound. Between the walls of the formerly mobile cartilage stopple and the bony rim of the fenestra a band of fibrous tissue was observed, which was evidently rendering the stopple less mobile or immobile for air-borne sound, its mobility varying with the postoperative period observed.

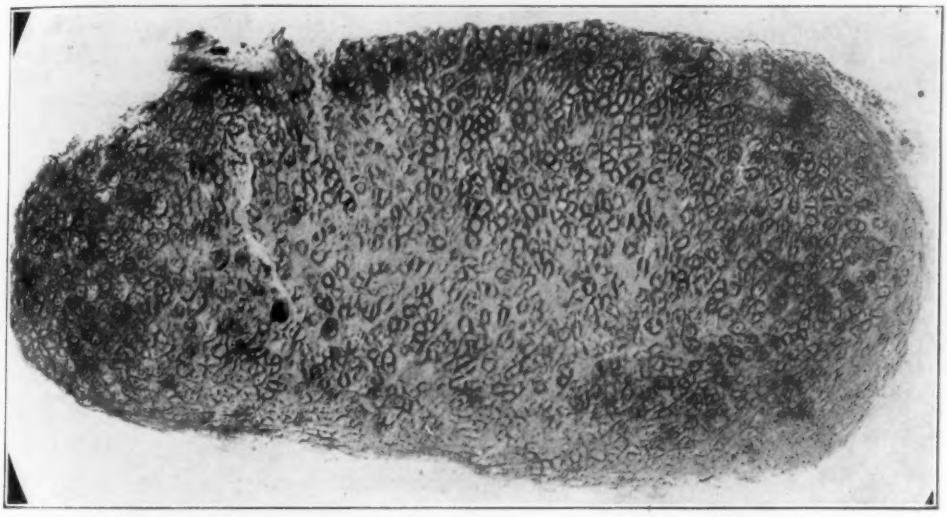

Fig. 14.—Stopple removed from a patient's fenestra when this was revised thirteen months after its creation.

In my article "Lempert Fenestra Nov-Ovalis with Mobile Stopple"[6] I stated:

> The mobile cartilage stopple simulates functionally the normal stapedial footplate.

I further stated:

> It is not unlikely, and further investigation may prove, that a fibrous structure simulating in function the annular ligament is eventually formed between the cartilage stopple and the bony rim of the fenestra.

It was the fact that this prediction came true, but not exactly as I wished it to, that resulted in a new problem to be solved. A fibrous structure did form between the cartilage stopple and the bony rim of the fenestra as predicted, but its function unfortunately is not like that

of the annular ligament—just the opposite. It gradually fixes the cartilage stopple, which was hitherto mobilizable by air-borne sound, and renders it immobile.

The next problem to be solved was how to employ a cartilage stopple so that it would prevent osteogenesis and also prevent the formation of a fibrous connective tissue band, so that the improvement of hearing obtained as a result of fenestration would be permanently maintained. From observations made in human subjects and a study of the histologic data obtained as a result of our scientifically controlled experiments with the cartilage stopple in the rhesus monkey it soon became obvious that when a cartilage stopple is inserted tightly instead of loosely into the fenestra osteogenesis is prevented and a connective tissue band does not form (fig. 15).

Unfortunately, however, the tight insertion of a cartilage stopple will not permit postoperative improvement of hearing, because, instead of acting as a mobile stopple and remaining mobilizable by air-borne sound, it functions as an immobilizable plug.

After trying the use of an immobilizable perforated stopple in a small series of cases I was forced to abandon it despite the knowledge that the formation of a connective tissue band could thus be avoided, because the hearing improvement obtained in these cases, though audiometrically significant, did not reach the level necessary for practical, serviceable unaided hearing. The reason for this was obviously the fact that the maximum-sized perforation which a cartilage stopple will permit to remain open while tightly hugging the bony rim of the fenestra is evidently not sufficient to permit the perilymph and the endolymph to be mobilized by air-borne sound to the degree necessary to raise the hearing to the practical level after the tympanomeatal membrane is placed to cover and seal the perforated stopple.

It soon became obvious that the only hope of reaching the desired objective with the use of the cartilage stopple lay in an investigation of the possibility and practicability of employing the immobilizable cartilage stopple in a two stage technic, the first stage consisting in forcing the healing without osteogenesis by tightly inserting the stopple, and the second stage consisting in establishing practical hearing by removing the stopple and sealing the healed fenestra with a tympanomeatal flap. That such a two stage procedure was feasible can be seen from the result obtained with this two stage technic in a boy 16 years old (fig. 16).

In my attempt to prevent osteogenesis according to the second principle—by devitalizing and inactivating the histologic structure responsible for it—I tried creating a highly polished surface in the three histologic layers of the freshly cut bony walls that form the rim of the newly created fenestra.

Fig. 15.—*A*, fenestra made in the left ear of monkey 37 in experiment III. At this level the cartilage stopple fits firmly into the fenestrated area. No regeneration of bone has occurred at this level. Note the piece of mature bone that has been shoved through the window by the stopple.

B, right ear of monkey 37 (control, in which no stopple was inserted) as it appeared three months after operation. Osteogenetic repair of the fenestra is almost complete.

In the July 1938 issue of the ARCHIVES OF OTOLARYNGOLOGY I [1] stated:

> An electrically driven no. 2 round dental finishing or polishing burr, the width of whose circumference is about one-half that of the outer surface of the external semicircular canal, is employed for the excavation of the trough.

In the May 1940 issue of the ARCHIVES OF OTOLARYNGOLOGY I [3] further stated:

> . . . After this [referring to the creation of the fenestra with the dental polishing or plug-finishing burr], the lateral bony walls of the trough are carefully burnished with a 24 carat gold burnishing burr. The purpose of employing the 24 carat gold burnishing burr is to reduce still further the possibility of bone regeneration in the occasional case by highly polishing the lateral bony walls of the fenestra and simultaneously implanting fine particles of gold within these walls.

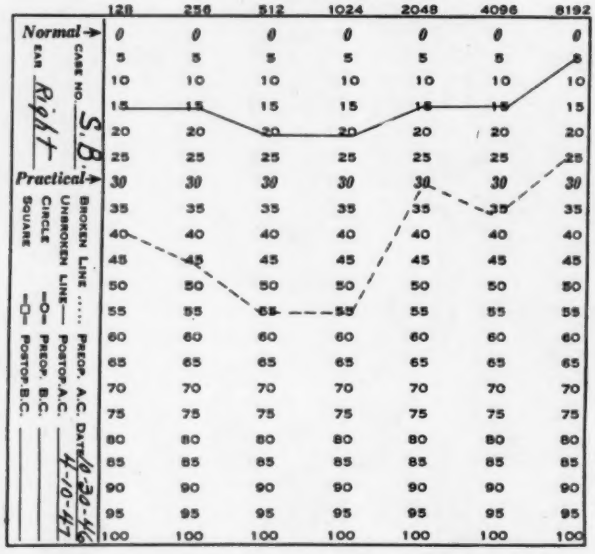

Fig. 16.—Audiogram of S. B., showing the successful outcome of the two stage technic for creating the fenestra nov-ovalis, in which a stopple is tightly fitted into the fenestra in the first stage, to force healing without osteogenesis, and is removed in the second stage, to establish serviceable hearing.

Though, as a result of scientifically controlled animal experimentation, reported in the October 1942 issue of the ARCHIVES OF OTOLARYNGOLOGY, Drs. Meltzer, Spector and myself [7] were able to observe histologically that the use of the dental polishing and gold burnishing burrs will intimidate and delay bone regeneration, there is no existing controlled and repeatedly proved histologic evidence that highly polishing the rim with these burrs will consistently prevent osteogenesis. The one and only

7. Lempert, J.; Meltzer, P. E., and Spector, B.: Bone Repair Following Defects Made in the Skull of Cats by Means of Different Instruments, Arch. Otolaryng. **36**:473-490 (Oct.) 1942.

histologic specimen obtained from human subjects, a photomicrograph of which was published in the May 1940 issue of the ARCHIVES OF OTOLARYNGOLOGY,[8] suggests that the polishing of the bony walls of the fenestra with the steel polishing and gold burnishing burrs did intimidate and delay osteogenesis (fig. 17). Since this specimen was obtained only two weeks after operation there is no way of knowing whether ultimately bone regeneration would not have taken place as it did in many other cases in which the steel polishing and gold burnishing burrs were employed to create the fenestra.

Fig. 17.—Highly polished periosteal, enchondral and endosteal bony layers of the fenestrated labyrinthine wall.

My attempts to prevent bone regeneration by devitalizing and inactivating the histologic structure of the freshly cut bony walls of the fenestra were not just limited to burnishing with the gold burnishing burr. I burnished variously in different series of cases with a silver burnishing burr, a steel burnishing burr and a pure lead burnishing burr and with silver amalgam.[6]

Through the courtesy of Dr. LeRoy Schall and under his auspices at the Harvard Medical School I outlined and directed a scientifically

controlled experimental investigation of the possibility of locally preventing osteogenesis within the bony walls of a newly created vestibular fenestra in the rhesus monkey by burnishing the fenestral rim with the various agents which I had employed in the human fenestra. Dr. Philip Meltzer and I performed the animal fenestrations. Dr. Dorothy Wolff supervised the preparation of the tissues at the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary and the Lempert Institute of Otology and interpreted the histologic appearances.

Dr. Meltzer and I approached this research problem with neither pessimism nor overenthusiasm. We were not interested to prove that a fenestra newly created in the human ear almost always closes, nor were we bent on proving that such a fenestra almost always stays open, for there exists no clinical basis for any such exaggeration.

Every otologist has observed that in a series of consecutive cases the most carefully executed performances of the best available technic for creating a labyrinthine fenestra will result in fenestral patency in some and fenestral closure in others for reasons nonexplainable.

Our experimental results represent an impartial investigation of the possibility of disposing of the inherent uncertainty accompanying fenestration surgery by finding and devising nondisputable, definitely proved means of always preventing osteogenesis from taking place within the bony walls of a newly created fenestra and thus assuring the permanent patency of every one of the newly created labyrinthine fenestras.

We do not have to devise and promulgate theories in the interpretation of the histologic observations of fenestras which remained patent as a result of our animal experiments, to prove that patency is not a biologic accident, because we excluded the possibility of accidental findings. Since we employed controls in our experiments and since we avoided complicating our experimental study by never investigating more than one experimental factor which could possibly have some bearing on osteogenesis at one time, a comparative histologic study and analysis of the results obtained in both experimental and control ears of the same rhesus monkey was sufficient to be considered as acceptable evidence of either success or failure of the experiment in question without the necessity of venturing into questionable hypotheses and theories.

In a histologic study of the 32 temporal bones of 16 monkeys which were subjected to experimental attempts to prevent osteogenetic closure of the fenestra by local devitalizion and inactivation of the structures responsible for it, with use of the various burnishing agents which I previously employed in the human ears, the following observations were made. Bone regeneration took place in the newly created fenestras in 30 of the 32 temporal bones. All the 16 control fenestras which were not burnished and 14 experimental fenestras which were burnished closed either completely or partially as a result of formation of new bone.

Only the right experimental fenestra of monkey 27 and the right experimental fenestra of monkey 31 remained widely patent because bone regeneration did not take place at the rim of the fenestra. *These were the only fenestras in which the freshly cut bony walls were burnished with pure lead.*

The left fenestra in each of these 2 monkeys, which was not burnished with pure lead or anything else and which was acting as a control for the experimental right ear, was closed completely by new bone.

In this experiment both the right and the left fenestra of each of the 16 monkeys were left unsealed and were not covered by any membrane, in order to exclude the tympanomeatal membrane as a possible factor in the evaluation of the results obtained. Therefore any histologic alteration beyond the rim of the fenestra must not be regarded as significant in our experiment (figs. 18 and 19).

This controlled experiment with pure lead has since been repeated in 3 more monkeys, and the results are being awaited.

A result obtained under such scientific control assumes significance because it excludes from our conclusions errors resulting from unknown biologic factors existing in the individual monkey which could influence osteogenesis one way or another. Since in none of the 16 monkeys used for these various experiments was the tympanomeatal flap employed, all of the experimental and control fenestras being left uncovered, we have a right to assume that the only factor which could have prevented osteogenetic closure of the right fenestras in monkey 27 and monkey 31 was necessarily the burnishing of the bony walls of the fenestra with pure lead, since in every other surgical detail the technic employed in both ears of each of these 2 monkeys was exactly the same.

No such significance can be attached to any reports of an occasional fenestra having remained open in the rhesus monkey after noncontrolled experimentation. Such noncontrolled experimental findings must be considered accidental and unexplainable, and no conclusions can be drawn therefrom which would shed light and point to means and ways of keeping a fenestra open.

It is indeed gratifying to find that at last and for the first time in the history of otology a scientifically proved means of preventing bone regeneration in a newly created fenestra of the labyrinth of the rhesus monkey has been found.

Because of our histologic observations regarding the 2 lead-burnished fenestras of the rhesus monkeys, I now in retrospect cannot help but feel, although special significance was not attached to it before, that perhaps it was more than a coincidence that in the only 3 patients in whose cases I burnished the fenestral rim with pure lead prior to the initiation of my animal research osteogenesis did not take place and the fenestras have remained open ever since (fig. 20).

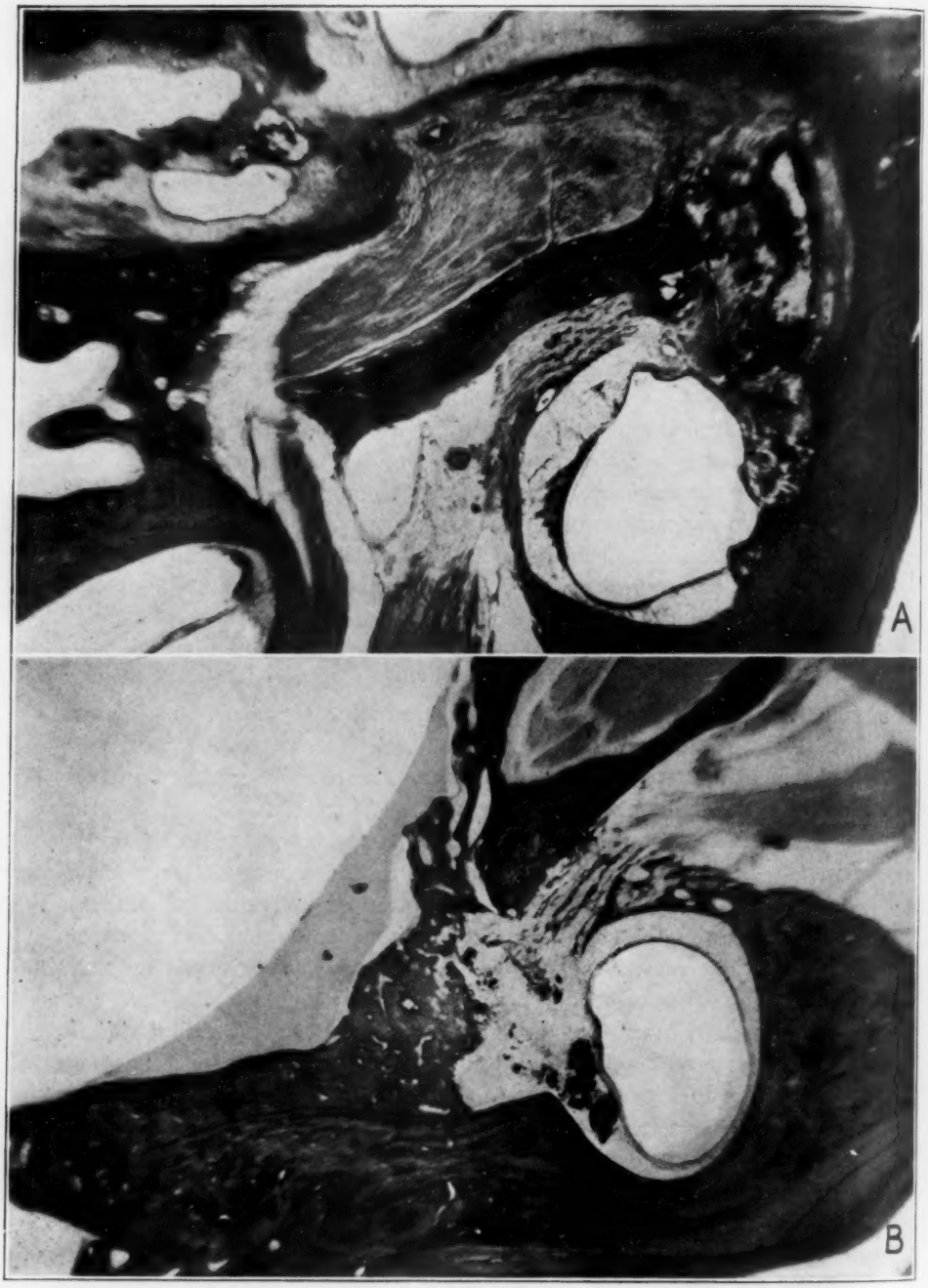

Fig. 18.—*A*, experimental fenestra burnished with pure lead (right ear of monkey 27, experiment II). No regeneration of bone is seen seven months after operation.

B, control fenestra, not burnished (left ear of monkey 27). The regeneration of bone is complete seven months after operation.

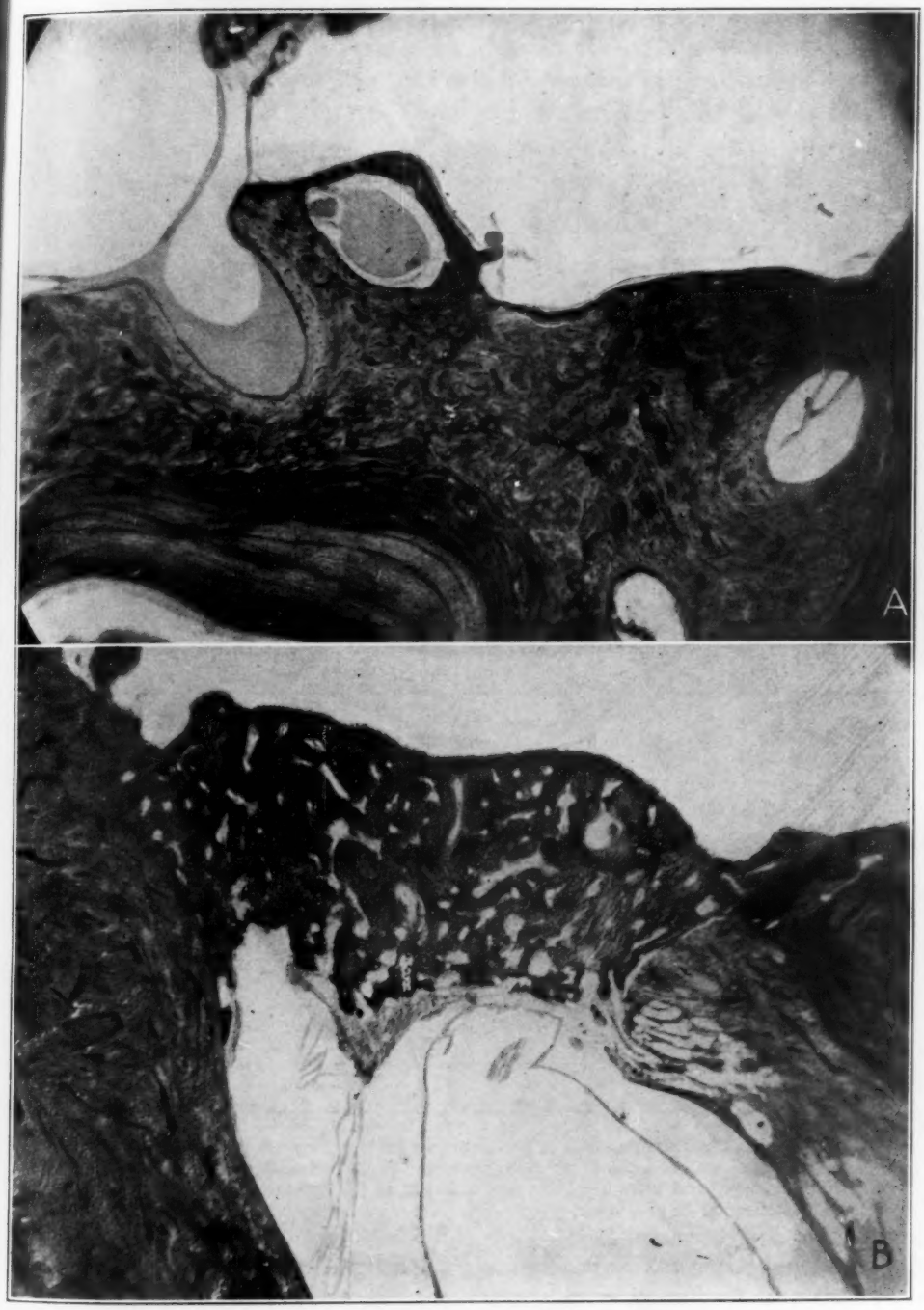

Fig. 19.—*A*, experimental fenestra burnished with pure lead (right ear of monkey 31, experiment II). No regeneration of bone is seen seven months after operation.

B, control fenestra, not burnished (left ear of monkey 31, experiment II). Repair of the fenestra is complete seven months after operation.

What is now being done to take advantage of what has to date been accomplished with our research in an attempt to advance the fenestra nov-ovalis from the individually nonpredictable to the individually predictable status?

In the hope that we may some day in the not too distant future be in a position always to prognosticate accurately whether or not the indi-

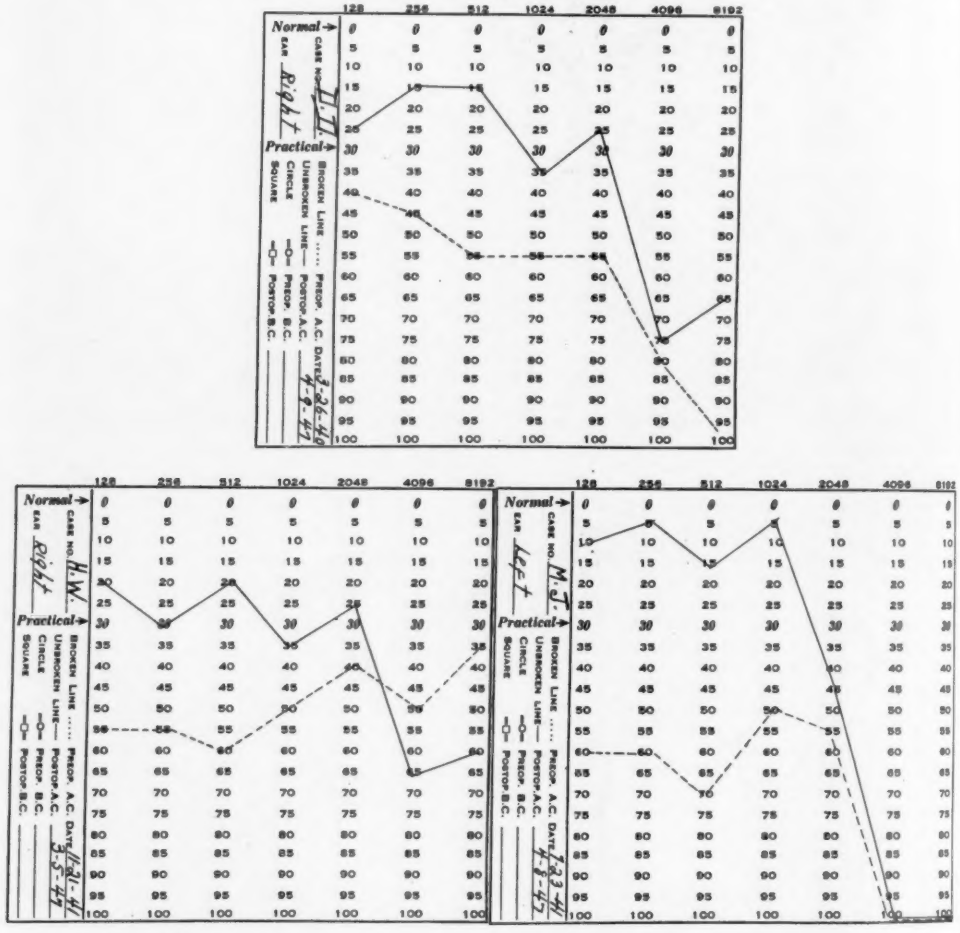

Fig. 20.—Audiograms of the 3 patients in whose cases alone the bony layers of the fenestral wall were polished with pure lead prior to animal research establishing that the pure lead finishing burr was a factor in the production of a fenestra free from formation of new bone.

vidual hearing can be restored to the practical, serviceable unaided level with the fenestra nov-ovalis operation, Drs. Wever, Lawrence, Meltzer and I are now investigating the possibility of developing a practical means of obtaining electrical cochleograms by which we may determine

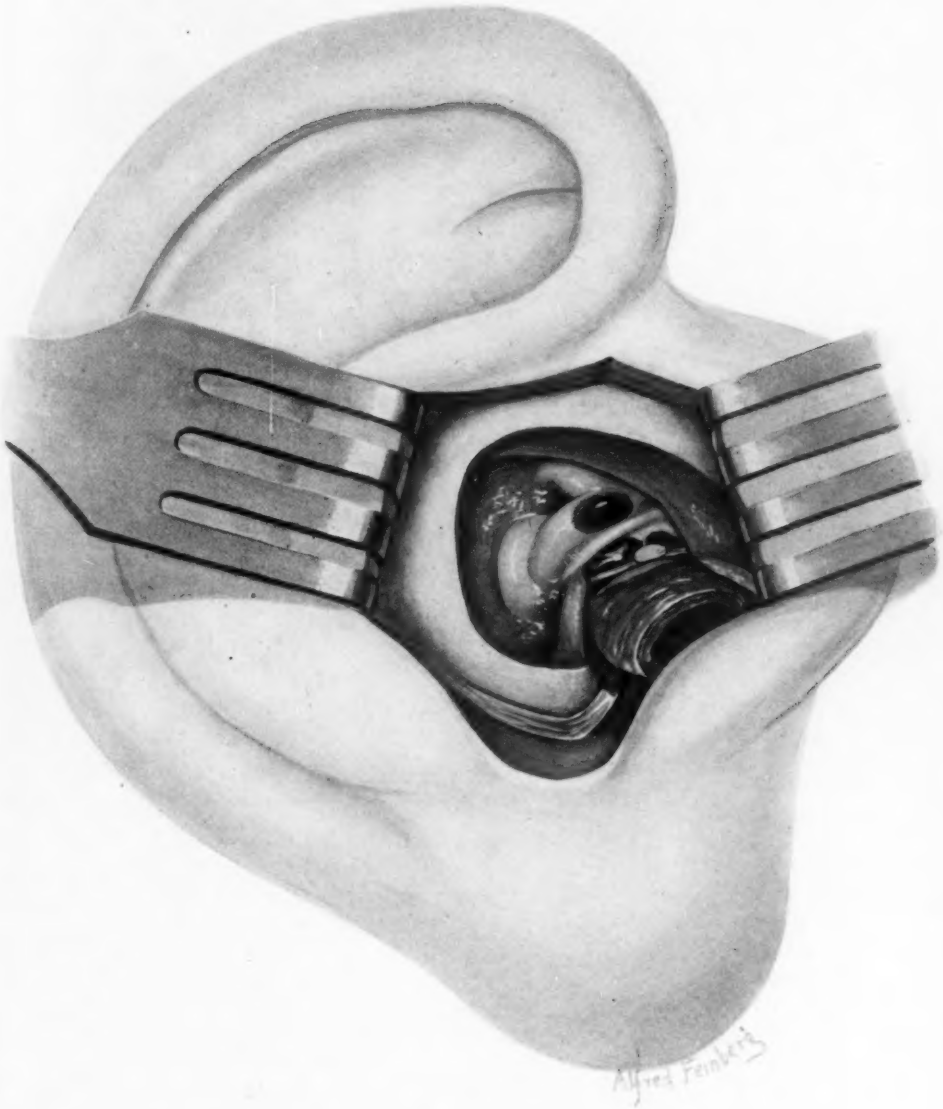

Fig. 21.—Drawing of a finished lead-burnished fenestra.

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preoperatively the existing degree of unused cochlear function in a deafened ear.

In furtherance of our ambition to be able to tell our prospective patient that when, after the fenestra nov-ovalis operation, his hearing is restored to the practical, serviceable level it will remain so as long as the existing cochlear nerve function remains unimpaired, I am now taking advantage of the knowledge gained from our histologic studies in the course of animal experimentation and applying it in my surgical treatment of the human subject.

On the basis of the histologic evidence obtained in our controlled animal experimentation that osteogenesis can be deliberately prevented from taking place in the freshly cut bony walls of a newly created labyrinthine vestibular fenestra of the rhesus monkey by burnishing the bony rim of the fenestra with pure lead, I am now attempting to reach in the human subject our long sought for objective, namely, the deliberate creation of a permanently patent fenestra which would insure the permanent maintenance of the practical, serviceable hearing obtainable postoperatively.

I am now continually employing the one stage lead technic in the hope that time will prove that all the surgical problems heretofore encountered in fenestration surgery can thus be finally and successfully solved.

LEAD-BURNISHED FENESTRA NOV-OVALIS

To prevent osteogenetic closure of the newly created fenestra and obtain a permanently patent fenestra nov-ovalis by locally devitalizing and inactivating the histologic structure responsible for the osteogenesis that arises within and spreads from the freshly injured bony walls of the fenestra I employ the following one stage lead technic:

STEP 1.—*Fenestration of the Bony Labyrinth.*—Only in the presence of an absolutely bloodless surgical field is a fenestra created within the bony capsule of the surgical dome of the labyrinthine vestibule. For this purpose an electrically driven 1.5 or 2 mm. steel plug finishing burr is employed, which is being constantly cooled with ice-cold isotonic solution of sodium chloride to prevent the high degree of postoperative inflammation of the bony walls of the fenestra that would be produced by the extreme heat of a noncooled electrically driven burr. The bone sand created by the burr is constantly and completely removed. The perilymphatic labyrinth is now exposed to view.

STEP 2.—*Fenestration of the Perilymphatic Labyrinth.*—With isotonic solution of sodium chloride, the pulverized endosteal bony layer of the labyrinthine capsule is carefully removed from the surface of the endosteum, which is the outer protective membrane of the perilymphatic labyrinth. The surface of the endosteal membrane is seen on the floor of the fenestra and may be intact or partly shredded. This membrane is now carefully floated with saline solution and gently teased outward and away from the underlying endolymphatic labyrinth with a no. 11 and 12 dental excavating instrument over the rim of the fenestra to the outer surface of the bony

labyrinthine capsule and thus removed. This when carefully done exposes to view the endolymphatic labyrinth in its normal position within the perilymphatic labyrinth.

STEP 3.—*Devitalization and Inactivation of the Freshly Injured Histologic Layers of the Bony Rim of the Fenestra with Pure Lead to Prevent Osteogenesis.*—The entire bony rim of the fenestra is thoroughly burnished with a 1.5 or 2 mm. lead burnishing burr, which is kept cool with ice-cold isotonic solution of sodium chloride, until lead particles are deposited in all its freshly injured histologic layers and turn grayish black. It is best to accomplish this burnishing slowly, by hand, instead of quickly, by electrically driven power. To protect the perilymphatic space from the occasional possibility that lead dust may enter it, though I never observed any ill effects therefrom when that happened, several sterile cotton fibers soaked in saline solution are placed over the presenting surface of the endolymphatic labyrinth and tucked under the edges of the endosteal bony layer of the fenestral rim. This cotton fiber floor, which may have gathered some lead dust, is removed as soon as the lead burnishing is completed, without danger of displacement or injury of the endolymphatic labyrinth.

STEP 4.—*Covering and Sealing of the Perilymphatic Space.*—The fenestra nov-ovalis is covered and sealed with the tympanomeatal membrane, which has been previously carefully prepared and fitted.

STEP 5.—*Promotion of Rapid Healing.*—The posterolateral bony wall of the postoperative cavity of the mastoid process, in which the exenteration of cells was limited to the absolute essentials of the technical requirements, is covered with a split thickness skin graft. This graft is obtained and prepared by utilizing the maximum amount of surplus skin obtainable from the posterior wall of the external auditory canal without in any way sacrificing either the sufficiency or the efficiency of the tympanomeatal membrane which has to be created in part from the cutaneous lining of the same wall. This has sharply reduced the postoperative healing period. With this step performed, the average postoperative cavity is completely healed and dry within a period of three to four weeks.

STEP 6.—The tympanomeatal membrane and the skin graft are held in the desired position by four carefully placed and firmly molded small pieces of paraffin gauze. The outermost two pieces of paraffin mesh are removed on the seventh postoperative day; the innermost two pieces are removed on the tenth day. This avoids the vertigo which usually followed the earlier removal of the packing.

COMMENT

From my experience with the lead-burnished fenestra nov-ovalis to date it would seem that:

1. Lead burnishing is evidently not injurious to the endolymphatic labyrinth or the organ of Corti. When we take into consideration also the fact that in 3 patients lead-burnished fenestras have now been functioning for more than five years and that the original hearing improvement has been maintained, we have definite evidence that lead in the infinitesimal quantity employed is certainly not injurious to the labyrinth.

2. The immediate results obtained so far with lead-burnished fenestras surpass the results obtained with the fenestras which were not lead burnished.

3. More time will have to pass before we can determine whether lead-burnishing of the rim of the fenestra nov-ovalis will prevent osteogenesis in the fenestra of the human subject as it did in that of the rhesus monkey and thus enable us to close the chapter of surgery for the improvement of hearing in patients with clinical otosclerosis finally and successfully.

SUMMARY

The evolutionary progressive efforts to achieve qualitative and quantitative permanent improvement of the end results of labyrinthine fenestrations in patients with clinical otosclerosis have been reviewed.

As a result of scientifically controlled experimentation done on the rhesus monkey, histologic evidence was obtained for the first time in the history of otology that bone regeneration can be deliberately and repeatedly prevented in newly created labryinthine fenestras by locally burnishing the bony rims with pure lead.

Without risking or endangering in the slightest degree the great progress already made and with every prospect and expectation of further enhancing the heretofore obtainable high percentage of good end results, I am now continually employing the lead-burnished fenestra nov-ovalis technic to prevent osteogenetic closure of the newly created fenestra in the patient, as it did in the rhesus monkey. If this objective is accomplished in the human being, heretofore individually nonpredictable status of the fenestration will be changed to the individually predictable status. Time will tell.

119 East Seventy-Fourth Street.

OSTEOGENESIS FOLLOWING FENESTRATION OF THE VESTIBULAR LABYRINTH OF THE RHESUS MONKEY

A Controlled Experimental Study

JULIUS LEMPERT, M.D.
NEW YORK
PHILIP E. MELTZER, M.D.
AND
LeROY A. SCHALL, M.D.
BOSTON
AND
DOROTHY WOLFF, Ph.D.
NEW YORK

THROUGH the courtesy of Dr. LeRoy A. Schall and under his auspices in the department of otolaryngology of the Harvard Medical School, Dr. Lempert outlined and directed a research investigation of the possibility of preventing osteogenetic closure of a fenestra newly created in the vestibule of the labyrinth of the rhesus monkey.

All the surgical operations with the exception of those of experiment II A were performed by Dr. Meltzer. The surgical operations of experiment IIA were performed by Drs. Lempert and Meltzer. The tissues were prepared in the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary and in the Lempert Institute of Otology, under the direction of Dr. Dorothy Wolff, who made the histologic reports.

The investigation was divided into three separate phases: Phase I was a study of the three individual histologic layers of the freshly cut bony rim of the newly created vestibular fenestra of the rhesus monkey, with regard to the part played by the periosteal, enchondral and endosteal layers in the process of osteogenetic repair of a newly created fenestra.

In phase II attempts were made to prevent osteogenesis from taking place in all the three histologic layers of the rim of a freshly cut fenestra by devitalizing and inactivating locally the histologic structures responsible for osteogenesis without altering systemically the existing inherent biologic factors governing osteogenesis in the rest of the body.

In phase III physical impedance was employed to prevent the start and spread of osteogenesis in the fenestral gap in order to force the healing of the freshly cut fenestral rim without osteogenetic closure of the fenestra.

In performing the experimental fenestrations, no attempt was made to protect the middle ear, the space containing the perilymph, the endolymphatic (membranous) labyrinth or the organ of Corti in any way at all. Therefore any histologic abnormalities observed in these regions have no bearing on our evaluation of the results of the experiments.

The experiments were limited to a study of how osteogenesis takes place in the freshly cut bony walls of the fenestra and to the investigation of the possibility of preventing osteogenesis in the region of the fenestral rim so that the fenestra could remain permanently patent.

In experiments I and II the fenestral gap was not sealed with a membrane; thus we disregarded what may happen as a result to the perilymphatic space and the endolymphatic structures within it. In order to control the experiments in question and remove doubt in the evaluation of the results, the possibility of the flap having to be considered as a factor in the explanation and interpretation of the resulting histologic observations had to be eliminated.

In experiments II and III, in which special experimental local factors were introduced in an attempt to prevent osteogenesis in the fenestral rim in the right ear of the rhesus monkey, the left ear was also operated on, the same technic as was used in the right ear being employed except that in the left ear the one single experimental factor used in the right for the prevention of osteogenesis was eliminated. Thus the right ear of each monkey was always the experimental ear, while the left ear acted as a control for the right experimental ear, except in monkey 37 in experiment III, in which the reverse was the case. Both ears were operated on at the same time except in monkeys 37, 41 and 7 in experiment III.

With such controlled experimentation it was possible in the evaluation of the end results to exclude error resulting from the fact that the constitutional biologic factors influencing osteogenesis in the individual monkey were unknown to us. With the controls employed, constitutional biologic factors need not be considered, since whatever their influence may be, that same influence affects osteogenesis in both ears.

EXPERIMENT I

Procedure.—Thirteen monkeys (26 ears) were used.

In this series of animals the endosteum was left intact in the right ear and removed in the left ear. The object was to observe and study osteogenesis in the freshly cut bony rim of the fenestra as to the manner and the time of onset of regeneration and repair and to observe whether the leaving or the removing of endosteum makes any difference in the end result, namely, the ultimate repair of the injury.

Results.—Thirty hours after the operation the fenestrated area was filled with free blood and fibrin (fig. 1 *A*).

TABLE 1.—*Experiment I*

Case	Monkey	Postoperative Period	Right: Experimental Ear	Result	Left: Control Ear	Result
1	64	30 hours (monkey died)	Endosteum intact	Free blood and fibrin in fenestra	Endosteum removed	Free blood and fibrin in fenestra
2	77	72 hours	Endosteum intact	Fibroblasts first appear, free blood, fibrin	Endosteum removed	Fibroblasts, very occasional; mostly free blood and fibrin
3	33	4 days	Endosteum partially intact	Early fibrosis	Endosteum removed	Fibroblasts present
4	11	6 days	Endosteum intact (some endosteal bone on it)	Fenestra filled with connective tissue	Endosteum removed	Fenestra covered with connective tissue
5	7	8 days	Endosteum intact	Fibrous tissue attached to microscopically thin layer of bone, "endosteum"		Free blood and fibrin within fenestra, connective tissue across lateral side of fenestra
6	12	10 days	Endosteum intact	Fenestra filled with connective tissue	Endosteum removed	Fenestra filled with connective tissue
7	13	12 days	Endosteum intact	Fenestra filled with connective tissue, periosteal bone	Endosteum removed	Fenestra filled with connective tissue; periosteal bone approaching fenestra
8	15	14 days	Endosteum intact	Regenerated trabeculae crossing fenestra	Endosteum removed	Islands of regenerating bone in fenestra; connective tissue
9	16	18 days	Endosteum intact; fenestra small	Bony trabeculae almost across fenestra	Endosteum removed; fenestra large	Islands of regenerating bone in fenestra
10	17 B (92)	22 days	Endosteum intact; fenestra in vestibule	Large quantity of mature bone fragments; new bone by accretion; some periosteal bone	Endosteum removed; fenestra in vestibule	Connective tissue and regenerating bone; large amount of mature bone fragments

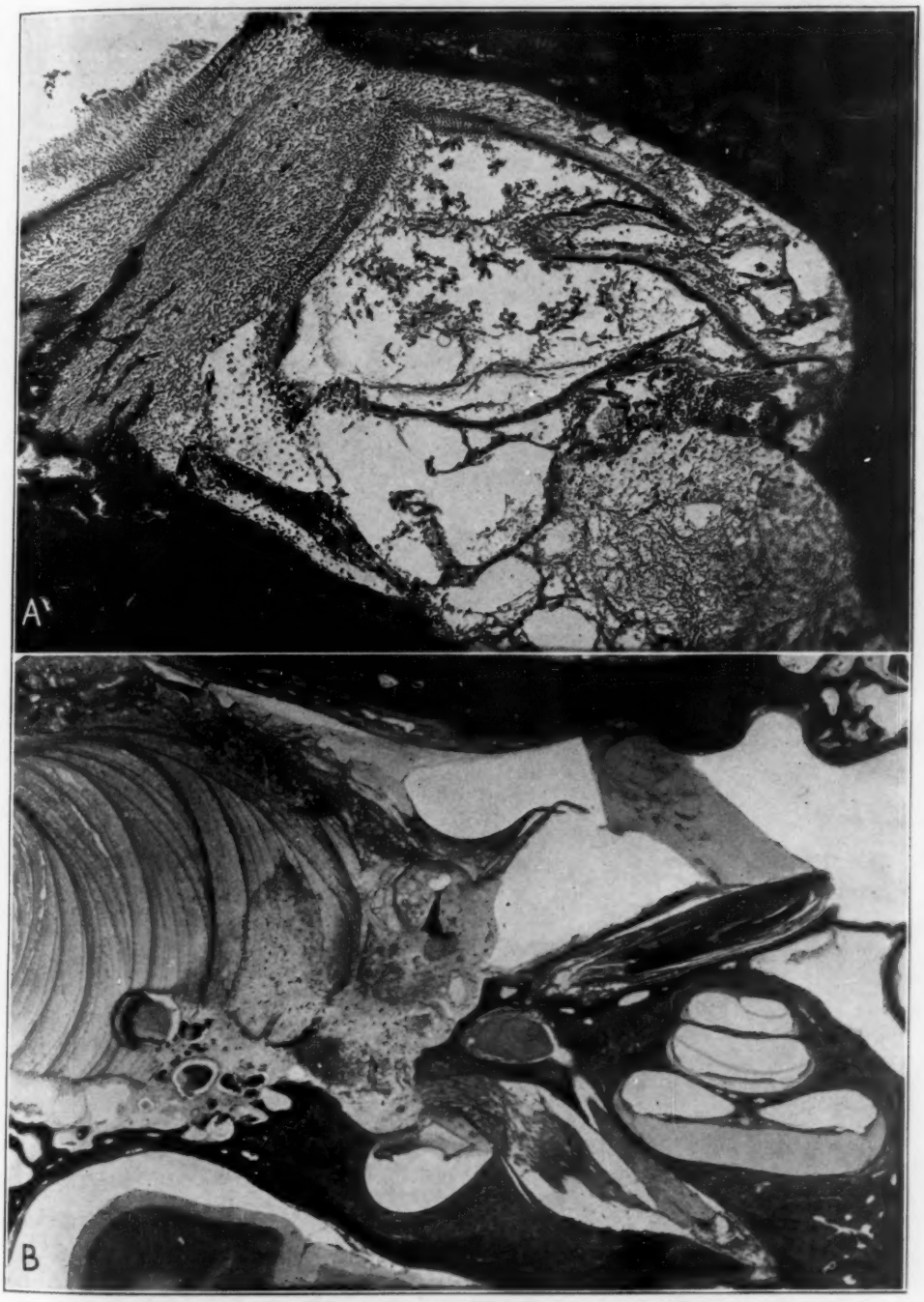

Fig. 1.—*A*, experiment I, monkey 64, left ear: endosteum removed. Free blood and fibrin are present within the fenestra nov-ovalis thirty hours after operation.

B, experiment II, monkey 11, left ear: endosteum removed. Granulations have occurred within the fenestra and are protruding into the tympanum six days after operation.

On the third and fourth days fibroblasts began to appear, but free blood and fibrin were still present.

On the sixth and eighth days connective tissue arising on the middle ear side had crossed the fenestrated area (figs. 1 *B* and 3 *A*).

On the tenth day connective tissue was not only over the fenestra but also within it, filling the space between the cut edges.

On the twelfth day periosteal bone was present, beginning to cross the window on the side where the endosteum was intact. New bone was just approaching the fenestrated area on the side on which endosteum had been removed (see Lempert's fig. 3 *A*, page 486).

On the fourteenth day islands of regenerating bone were observed in the fenestra on the side on which endosteum had been removed (see Lempert's fig. 4 *A*, page 487).

Fig. 2.—Experiment I, monkey 15, right ear. Periosteal bone is crossing the fenestra fourteen days after operation. The endosteum is in situ.

Bone cells freed from the cut edges of the bone were contributing fibroblastic processes to tissue filling in the window.

On the twenty-second day regenerating bone was arising not only from periosteum but, by accretion, from fragments of bone left in situ and from endosteum (see Lempert's fig. 4 *B*).

By the twenty-ninth day regeneration was complete.

Although early stages of fibrosis and regeneration showed more rapid development on the side on which endosteum had been left intact, later stages showed complete regeneration on both sides with no distinguishing difference.

No evidence was obtained that either the removal or the leaving intact of the endosteal membrane prevents osteogenesis from taking place.

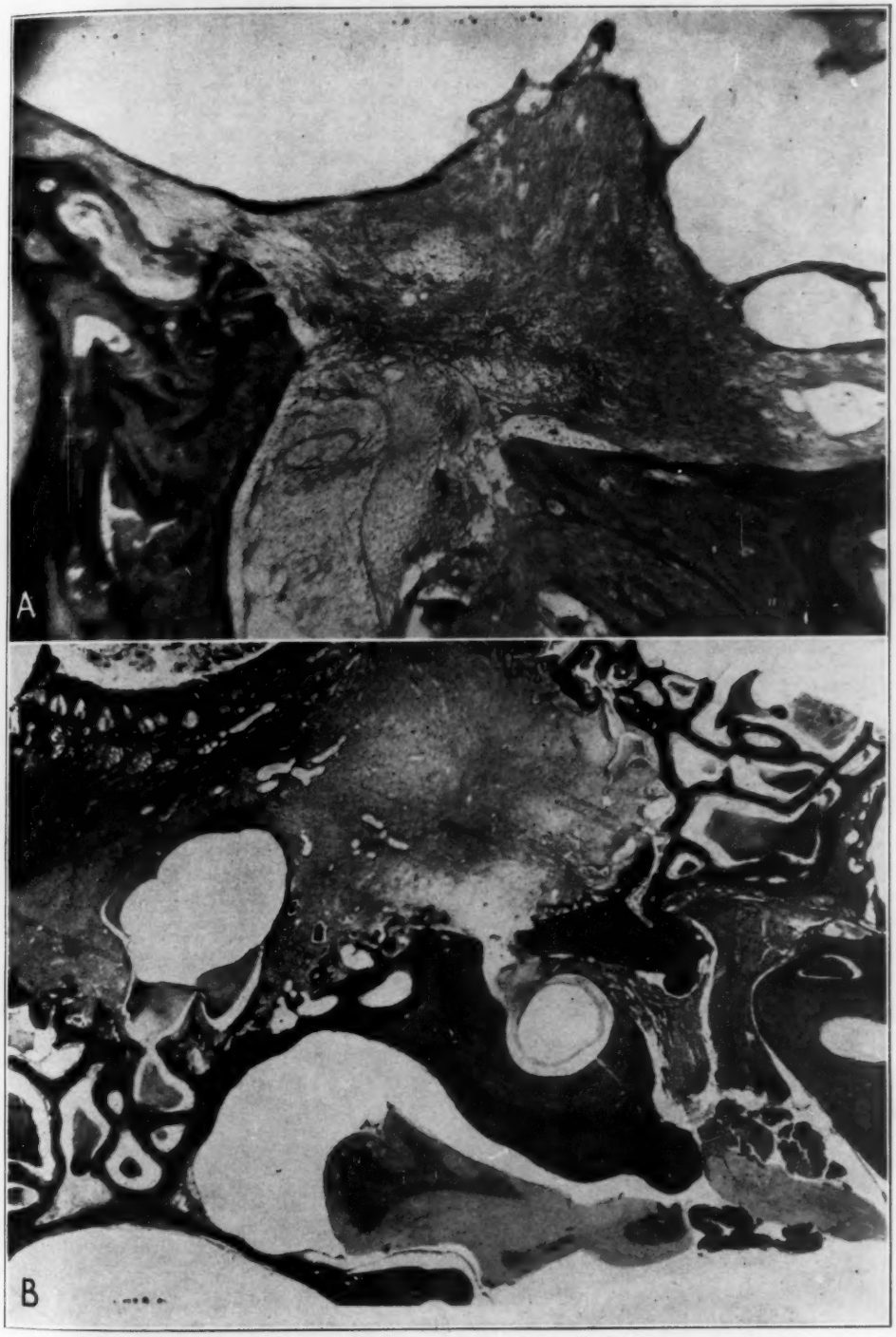

Fig. 3.—*A*, experiment I, monkey 7, left ear. Scar tissue plugs the fenestra eight days after operation.

B, experiment I, monkey 13, left ear. Periosteal bone is approaching the fenestra twelve days after operation. Endosteum was removed. Note that the whole middle ear is filled with scar tissue.

TABLE 2.—*Expermient II*

Case	Monkey	Postoperative Period	Right: Experimental Ear	Result	Left: Control Ear	Result
			A			
1	19	6 months	Polishing burr	Regeneration complete	Sharp cutting burr	Regeneration complete
2	28	3 months (monkey died)	Polishing burr	Regeneration complete	Sharp cutting burr	Regeneration complete
3	32	7 mo. 7 days	Polishing burr and electrocoagulation	Regeneration complete	Polishing burr only	Regeneration complete
4	24	8 mo. 2 weeks	Electrocoagulation	Regeneration complete	Polishing burr	Regeneration complete
5	34	7 mo. 7 days	Steel burnishing burr	Regeneration complete	Polishing burr	Regeneration complete
6	35	1 month (monkey died)	2% aqueous gentian violet	Widely patent with delicate connective tissue present	Polishing burr	Widely patent, with delicate areolar tissue; constitutional biologic factor?
7	21	6 weeks (monkey died)	Silver burnishing burr	Early regeneration	Polishing burr	Regeneration beginning; endosteum active
8	30	7 mo. 8 days	Silver burnishing burr	Regeneration complete	Polishing burr	Regeneration complete
9	25	8 days (monkey died)	Silver amalgam	No regeneration even of connective tissue	Polishing burr	Connective tissue only
10	26	8 mo. 2 days	Silver amalgam	Regeneration incomplete	Polishing burr	Regeneration complete
11	23	8 mo. 3 weeks	Gold burnishing burr	Regeneration complete	Polishing burr	Regeneration complete
12	29	7 mo. 5 days	Gold burnishing burr	Regeneration complete	Polishing burr	Regeneration complete
13	40	7 months	Gold burnishing burr; large fenestra	Regeneration complete	Polishing burr	Regeneration complete
14	36	6 months	2% aqueous gentian violet and gold burnishing burr	Regeneration complete	Polishing burr	Regeneration complete
15	27	6 mo. 28 days	Lead burnishing burr	No regeneration at fenestra	Polishing burr	Regeneration complete
16	31	6 mo. 27 days	Lead burnishing burr	No regeneration at fenestra	Polishing burr	Regeneration complete
			B			
1	20	6 months	Entire lateral canal exposed	Regeneration complete	Small fenestra	Regeneration complete

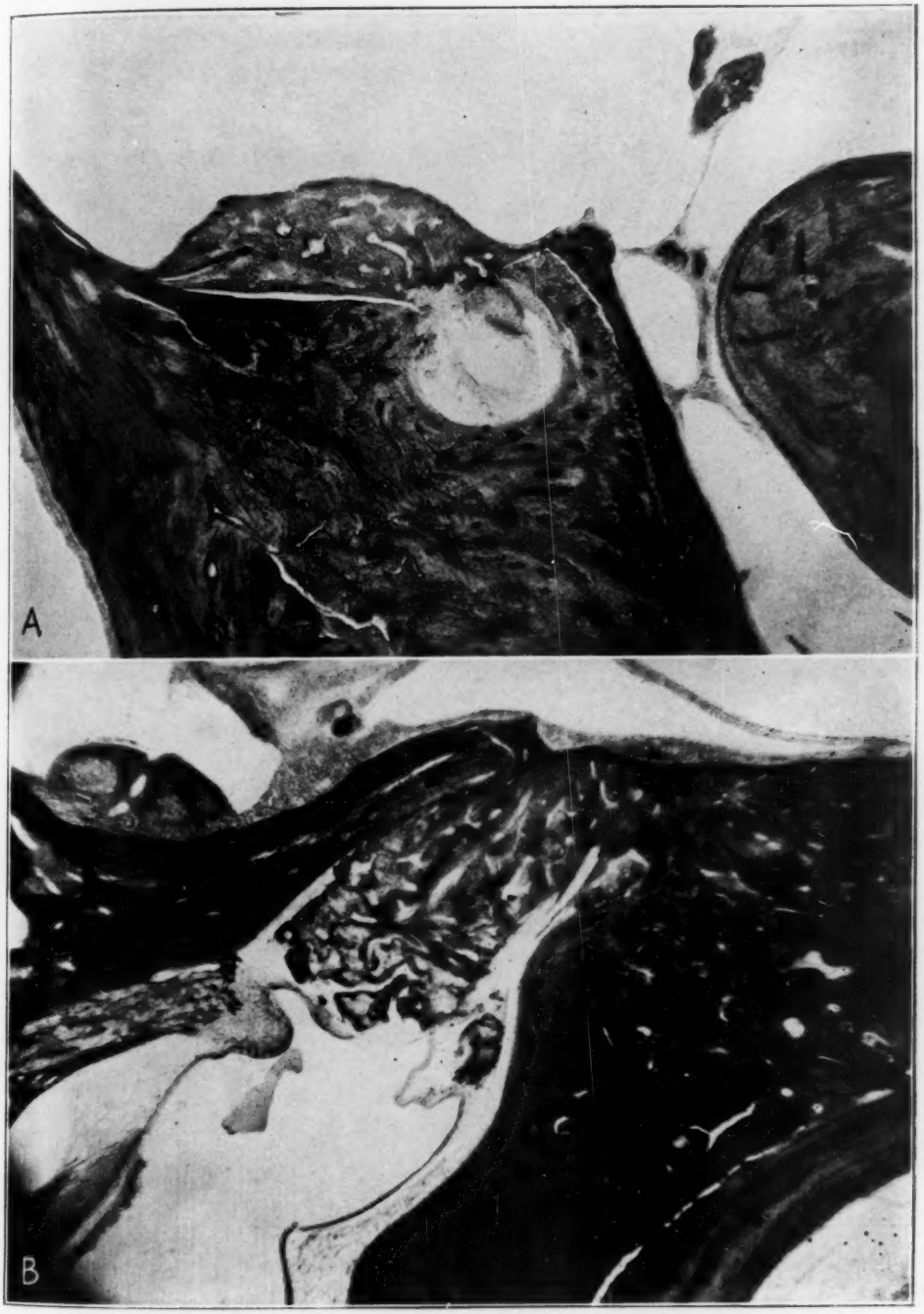

Fig. 4.—Experiment II, monkey 30. *A*, right, experimental ear, in which the fenestra was burnished with silver. Bone regeneration is complete seven months one week after operation.

B, left, control ear, in which the fenestra was not burnished. Regeneration of bone is complete seven months one week after operation.

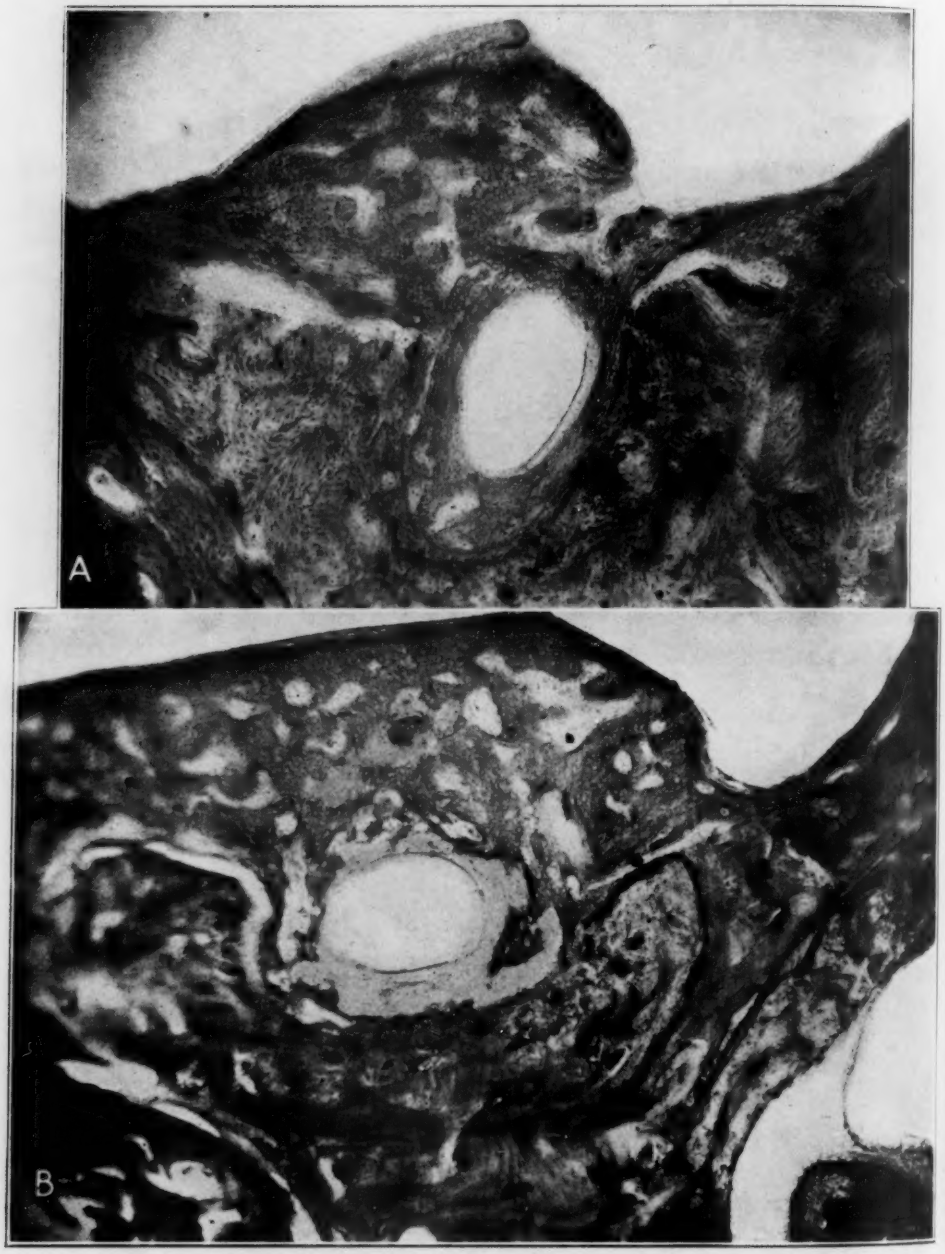

Fig. 5.—Experiment II, monkey 34. *A*, right ear, in which the fenestra was burnished with a stainless steel burr. Bone regeneration is complete seven months one week after operation.

B, left ear (control), in which the fenestra was not burnished. Bone regeneration is complete seven months one week after operation.

Fig. 6.—Experiment II, monkey 26. *A*, right ear, in which the fenestra was burnished with silver amalgam. Regeneration of bone is incomplete eight months after operation.

B, left ear (control), in which the fenestra was not burnished. Regeneration of bone is complete eight months after operation.

Fig. 7.—Experiment II, monkey 40. *A*, right ear, in which the fenestra was burnished with a 24 carat gold burr. Bone regeneration is complete seven months after operation. Note the protruding plug of new bone. Compare this with figure 3 *A*, in which a similar plug is seen in scar tissue.

B, left ear (control), in which the endosteum was left intact and the fenestra was not burnished with gold. Regeneration of bone is complete seven months after operation.

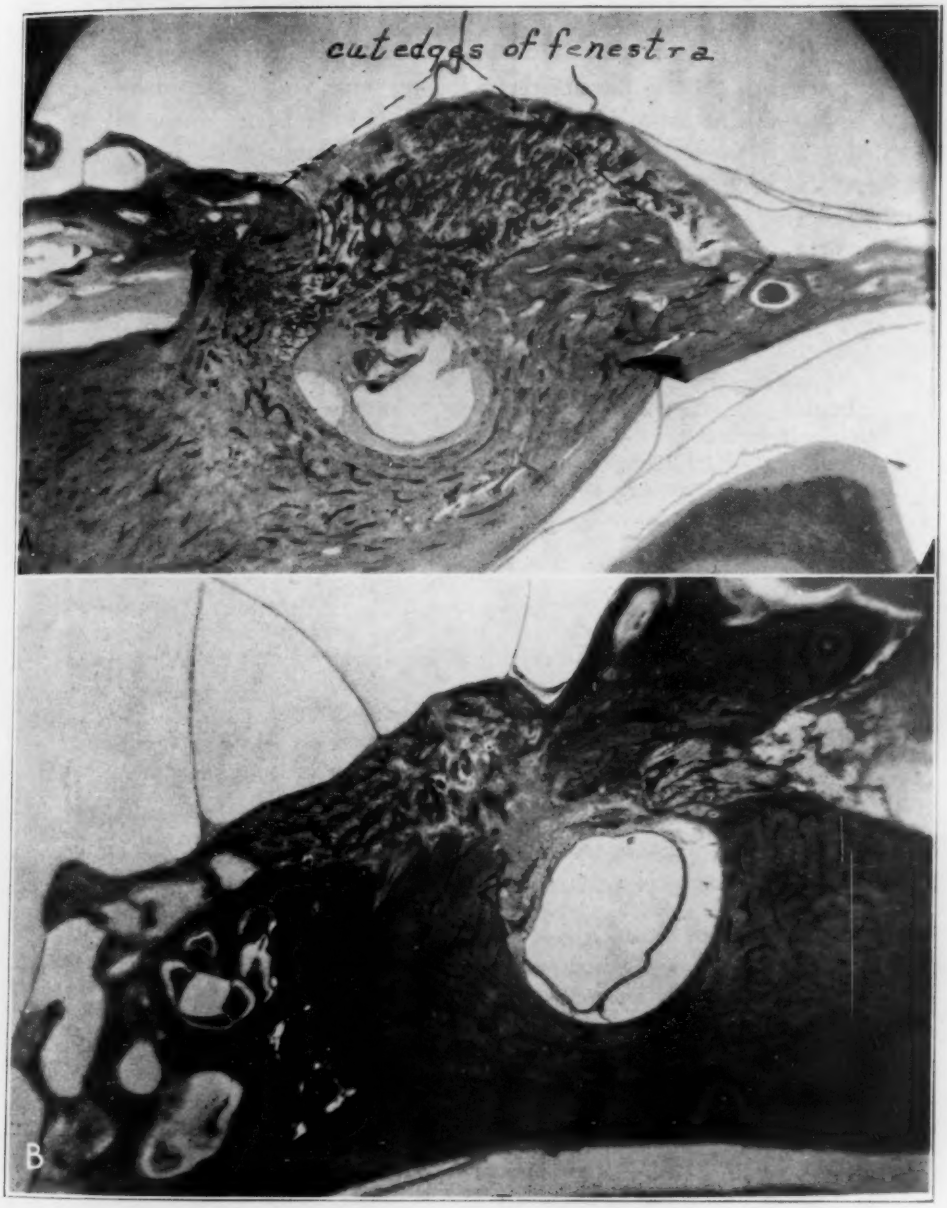

Fig. 8.—Experiment II, monkey 24. *A*, right ear in which electrocoagulation of the walls of the fenestra was tried. Regeneration of bone is complete eight months two weeks after operation.

B, left ear (control), in which there was no electrocoagulation of the walls of the fenestra. Regeneration is complete eight months two weeks after operation.

Comment.—Regeneration is initiated by fibrosis, which fills up the whole middle ear, crossing over the fenestra at first, later filling in the fenestra.

Periosteal bone is the first bone to regenerate in the untreated ear.

After this, more or less simultaneously endosteal bone, bone deposited by accretion around mature fragments and islands of the new bone, deposited independently, appear.

There is no existing evidence that enchondral bone is immune to regeneration. Enchondral bone was observed to regenerate in some of the experimental and control ears of the rhesus monkey and not in others.

EXPERIMENT II—A

Procedure.—Sixteen monkeys (32 ears) were used.

In this experiment attempts were made to inactivate the histologic elements responsible for osteogenesis by burnishing the freshly cut rim of the fenestra in the right ear of the rhesus monkey with various metals, with a medicament and with an electrocoagulating current. These burnishing agents had been employed in the human subject by Dr. Lempert prior to the initiation of this experiment. In the left ear of the rhesus monkey the experimental factor was not used and the fenestral rim was not burnished. Otherwise the technic was the same as in the right ear. The left was the control ear.

Results.—Burnishing with 24 carat gold did not prevent bone regeneration in the newly created fenestra. Bone repair was complete in both the experimental and the control ear.

Silver burnishing did not prevent bone regeneration.

Burnishing with silver amalgam retarded but did not prevent bone regeneration.

Burnishing with 2 per cent aqueous solution of gentian violet retarded but did not prevent regeneration of bone.

Burnishing with stainless steel did not prevent bone regeneration.

Burnishing with electrocoagulating current did not prevent bone regeneration.

Burnishing with pure lead prevented bone regeneration in the newly created fenestra after six months twenty-eight days in the only 2 monkeys in whose cases the experimental fenestra was burnished with pure lead. The control fenestras of these 2 monkeys were completely repaired by new bone (see Lempert's figs. 18 and 19, pages 506 and 507).

EXPERIMENT II—B

Procedure.—In the right ear of monkey 20 the entire external semicircular canal was fenestrated. In the left ear a small fenestra was made in the external semicircular canal.

Result.—Regeneration was complete in both ears.

Comment.—Length of fenestra seems to have no bearing on ultimate regeneration of bone.

EXPERIMENT III

Procedure.—Six monkeys (12 ears) were used.

A cartilage stopple was placed in the fenestra of the experimental ear. Over this a flap was placed. In the opposite or control ear the flap was placed over the fenestra but no cartilage stopple was used.

TABLE 3.—*Experiment III*

Case	Monkey	Postoperative Period	Right: Experimental Ear	Result	Left: Control Ear	Result
1	37	R: 3 mo. L: 3 mo. 25 days	Membranous flap; 2 × 1 mm. fenestra; paraffin	Regeneration almost complete	Cartilage stopple from inferior margins of helix; flap; paraffin	No regeneration where stopple fitted firmly, but regeneration above and below
2	41	R: 3 mo. L: 3 mo. 2 weeks	2.5 × 1 mm. fenestra; cartilage stopple from tragus; flap; paraffin	Regeneration around stopple filling in rest of canal	Labyrinth damaged; plastic flap; paraffin	Regeneration complete
3	42	6 mo. 1 week	Cartilage stopple from tragus; flap; paraffin	Fenestral bone not completely regenerated; flap in situ	Thiersch graft (skin graft)	Graft in situ; no regeneration here
4	44	4 mo. 1 week	Cartilage from helix; no flap; paraffin; experiment considered a failure	Regenerated bone anchoring stopple	Cartilage from tragus; flap, a separate skin graft	Cartilage displaced inward, surrounded by bone
5	45	5 mo. (monkey died, cause unknown)	Cartilage; flap; paraffin	Bone regeneration over surface of cartilage where stopple is depressed	Flap over small fenestra	Regeneration; small cholesteatoma
6	7	R: 6 mo. 24 days L: 4 mo. 19 days	Cartilage stopple from tragus; flap; paraffin	Stopple invaded by bone	Flap only	Regeneration almost complete; flap in situ
7	16	4 mo. 10 days	Cartilage stopple from tragus; flap not maintained	Regeneration around, not over, cartilage	Flap only	Regeneration complete

TABLE 4.—*Experiment IV*

Case	Monkey	Postoperative Period	Right: Experimental Ear	Result	Left: Control Ear	Result
1	47 (Linden)	4 mo. 2 weeks	Laminated fibrin stopple, no. 665	Regeneration complete	Plastic film no. 61	No diagnosis; technically too poor
2	48 (Linden)	3 months	Laminated fibrin film	Regeneration complete	Laminated fibrin film	Regeneration; technically very poor
3	55 (Linden)	2½ months	Plastic fibrin film; paraffin	Regeneration complete, technically poor	Fibrin film flap; paraffin	Connective tissue and new bone; technically poor
4	59	6 mo. 1 week	Fibrin film, type used by neurosurgeon for dura substitute, laid over fenestra; retained with fibrin foam; cavity packed with fibrin foam	Regeneration	Skin of canal placed over fenestra; retained with paraffin	Periosteal bone occludes fenestra
5	46	3 mo. 2 weeks	Fibrin film; blood clot allowed to fill cavity; uncovered	Endosteal bone completely closed fenestra; canal invaded	Fibrin film; flap used	Regeneration complete

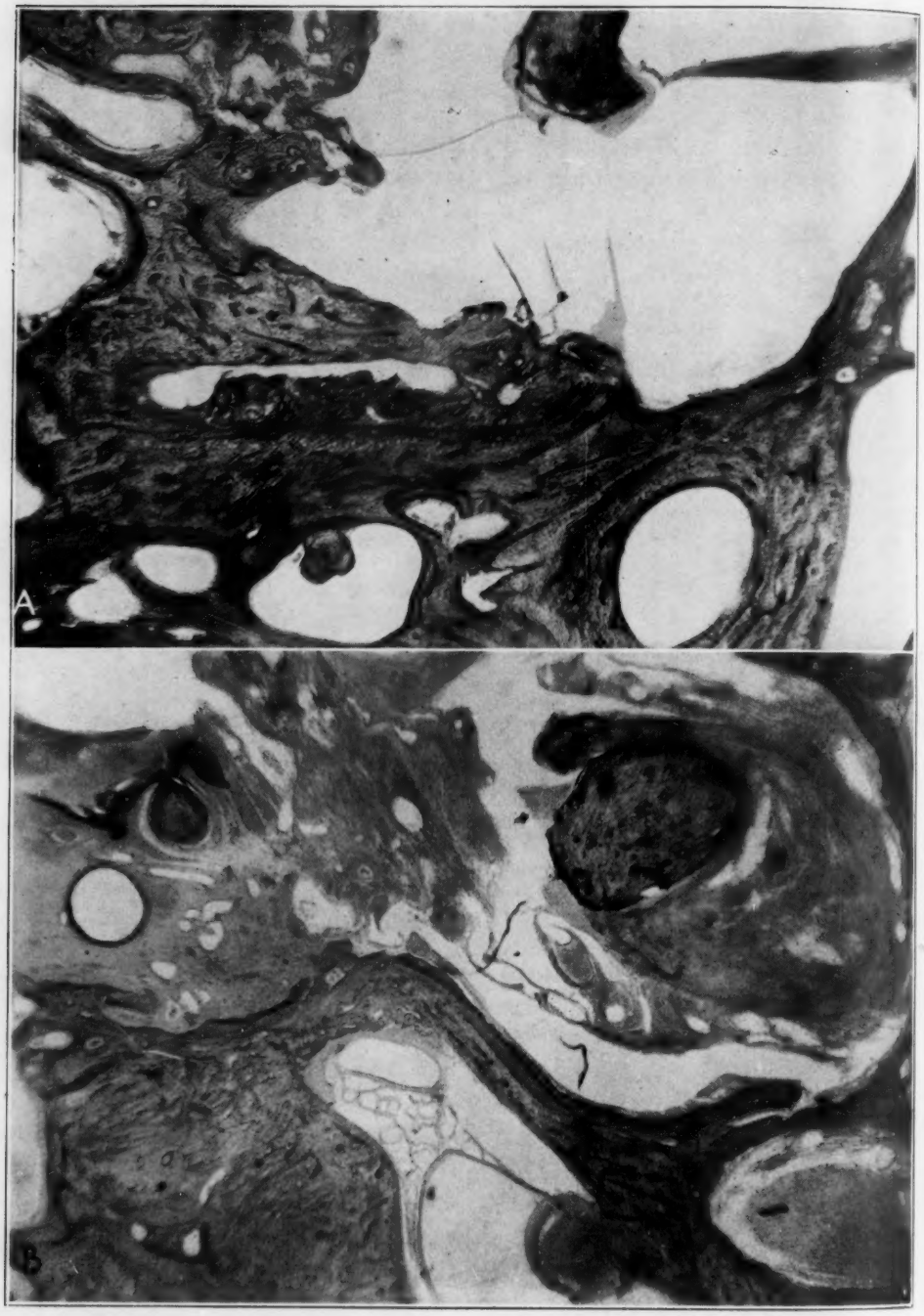

Fig. 9.—Experiment III, monkey 46. *A*, right ear, in which fibrin film was placed in the fenestra. Three months two weeks after operation repair of the window is complete, the newly formed bone apparently rising from endosteum. *B*, left ear (control), in which a flap was placed over the fibrin film that was inserted into the fenestra. Note the complete closure of the fenestra with new bone three months two weeks after operation.

Results.—Examination of the experimental ears showed that after three months the cartilage stopple was usually firmly anchored by new bone filling in above and below the level of the stopple. New bone also may invade the cartilage stopple. Regeneration did not occur where the stopple fitted firmly the cut edge of the fenestra.

Comment.—Regeneration did not occur where the cartilage stopple fitted firmly against the cut edge of the capsule (see Lempert's fig 15, page 501.).

EXPERIMENT IV

Procedure.—Five monkeys (10 ears) were used.

Fibrin film was inserted into the fenestra of the experimental ear. In the control ear, either a different kind of fibrin film or the skin flap alone was used.

Results.—In monkeys 59, 55 and 46 plastic fibrin film was placed in each fenestra. In the left ear the flap was used. In the right, no flap was used.

In monkey 47 two different kinds of plastic film were inserted within the fenestra. No tympanomeatal flap was used on either side.

Regeneration occurred beginning as early as two and a half months after the operation. All experimental and control fenestras in this series showed complete regeneration.

Comment.—Fibrin foam or film does not prevent regeneration.

After examination of all the ears in which flaps were used, it must be said that the presence of the skin flap over the window does not prevent regeneration of endosteal bone from occurring beneath the flap or even periosteal bone from creeping under the flap and crossing the window.

SUMMARY

Osteogenetic repair of a newly created labyrinthine vestibular fenestra of the rhesus monkey and its prevention were studied. To permit proper evaluation of the results, all the experiments were scientifically controlled. Both ears of each monkey were always operated on; the right ear was always the experimental ear and the left ear was always the control ear except in monkey 37 in experiment III, in which the reverse was the case.

Eighty-two ears of 41 monkeys were operated on. As a result of these experiments it was observed that bone regeneration took place in the fenestral regions of both ears of every monkey operated on with the exception of monkey 27 and monkey 31, the only monkeys in which the experimental right fenestra was burnished with pure lead. In these 2 monkeys, the experimental right fenestra, which was burnished with pure lead, remained patent, showing no evidence of bone regeneration, whereas the control left fenestra, which was not burnished with pure lead, closed by bone regeneration.

119 East Seventy-Fourth Street.

REVISION OF THE FENESTRATION OPERATION

PHILIP E. MELTZER, M.D.
BOSTON

THIS subject has been little discussed in the recent literature, and now that otologists have had the opportunity of reviewing statistical reports of the end results of fenestration operations, it becomes apparent that they are definitely in need of such a discussion. The more experienced surgeons are agreed that when cases are well selected a large majority of the patients will have their hearing improved to the practical level in consequence of the fenestration operation. The improvement occurs usually within three to six weeks. The percentage of cases in which hearing is so improved varies, according to the reports, from 70 to as high as 90 per cent. These figures are strikingly impressive. Whatever the actual percentage of early good results may be, ultimate failures are of such frequency that the question of revision becomes one of major importance.

REASONS FOR FAILURE TO OBTAIN AND MAINTAIN IMPROVEMENTS OF HEARING

Accidents excluded, it becomes apparent that osteogenesis, fibrosis and labyrinthitis, in this order of importance, are the causes of postoperative failure. Labyrinthitis due to infection is a negligible factor. The evidence of an existing labyrinthitis is definite and unmistakable. When the labyrinthitis is mild, the hearing of the higher frequencies only may be impaired; that of the lower frequencies, not at all or slightly impaired. When the labyrinthitis is severe, the hearing of all frequencies drops below the preoperative level. Mild labyrinthitis ends in recovery frequently; the severe forms, much less frequently. Some labyrinths apparently can withstand considerable trauma.

Fibrosis as a cause of failure is difficult to evaluate clinically. When the membranocutaneous canal wall flap is laid down over the fenestra, careful as one may be, a film of organized blood elements will accumulate under it. This matrix becomes fibrous tissue and binds the flap to the bony capsule. In some cases, for unknown reasons, the fibrous tissue grows into the window and partly into the space containing the perilymph. It may completely obliterate the perilymphatic space. The fact that the majority of patients do not obtain good improvement of hear-

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ing indicates that the blood elements for the most part become absorbed from within the fenestra and the perilymphatic space.

The most important cause of closure of the fenestra is osteogenesis, and the endosteum and the periosteum are the tissues in which this activity begins. The new bone may completely or only partially replace the connective tissue matrix. It may do so rapidly or slowly. It may pile up in an exuberant growth, rough and knoblike, at the site of the fenestra. It may begin at the margin of the fenestra and grow concentrically toward the center, never completely closing the window. New bone may grow into the perilymphatic space. Usually, when regeneration occurs, the new bone restores the contour of the osseous labyrinth. Sometimes it fails to do so, and one sees a saucer-like depression in the fenestra. The new bone is easily distinguishable in the first year or two; later it may become indistinguishable from the surrounding bone.

It is possible for any combination of the aforementioned causes of failure to take place to a greater or a lesser degree, and it may result in early or late loss of the postoperative improvement of hearing.

Up to the time of Lempert's announcement of his procedure the hearing improvement following operation was almost invariably lost because of closure of the fenestra. This occurred, regardless of the technic employed. For this reason the magnitude of his contribution to the surgical treatment of otosclerosis cannot be overemphasized. Apparently, in the majority of patients the Lempert technic does something which inhibits the reparative powers of the tissues necessary for the healing of the fenestra. Just what these factors are is not known. Otherwise, how can one account for the failures wherein every detail of his technic was adhered to. Meticulous observance of technic, important as it may be, and though it will undoubtedly improve the percentage of good results, does not supply the answer to the question whether the fenestra will remain open and the improvement of hearing be maintained.

The anxious waiting of patient and surgeon following the operation is one of the distressing features of this work. Many patients are doomed to disappointment because of their remarkable power of tissue repair. This variable power is dependent on constitutional factors beyond one's control. Some surgeons doing this work have not been able to obtain the high percentage of success which has been reported. To them, revision is important, and they would like to know when and under what circumstances reoperation is a reasonable undertaking.

Lempert's report of observations made in revisions [1] will remain one of the fundamental pioneer contributions on this subject. Those early

1. Lempert, J.: Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis: Summary Report of One Hundred and Twenty Cases, Arch. Otolaryng. **31**:711 (May) 1940.

revisions were the foundation for his newer advanced modification of the technic.

SELECTION OF THE PATIENT FOR REVISION

Suitability is determined after a complete study has been made of the record of the patient. This should include preoperative audiograms, a review of what occurred at the original operation and a review of what transpired after the operation.

Assume that the operative procedure was carried out perfectly; the fenestra was well made, approximately 4 to 4.5 mm. long and 1.5 mm. wide, with no endosteal shreds and with bone debris and chips thoroughly removed from the cavity. Such being the case, I offer the following opinions as to when revisions should or should not be done.

First: When not to revise:

1. A revision should not be undertaken in a patient who fails to get any improvement of hearing following the operation.

2. It should not be done in a patient whose hearing recedes after he has obtained a limited improvement only. The hearing which never reaches the practical level at any time postoperatively should not be taken to indicate revision.

3. Revision is not to be done in a patient whose threshold is improved considerably in the lower frequencies but not to the practical level in the important speech frequencies.

4. It is unwise to reoperate on a patient who had a good improvement and then rapidly lost it within a matter of three to six weeks, especially if the hearing has receded below the preoperative level. This usually means fibrosis.

Second: When to revise:

1. A revision may be undertaken in any case in which inexperience and temerity resulted in the creation of too small a fenestra. In cases of this type the patient may lose his improvement rather rapidly, within six to eight weeks.

2. If the hearing improvement remains for six to eight weeks and is then rapidly lost and one has a feeling that a fragment of bone might have made its way into the window or that possibly a spicule of bone was attached to the canal wall membrane, one is justified in revising sooner than usual, provided the cavity is clean and well epidermized.

An example of a type of case ideal for revision would be one in which the audiogram made at the first operation showed for air conduction a linear loss at about 50 decibels in all frequencies, with excellent bone conduction, as tested by forks and audiometer, and in which, after the operation, the following sequence was presented: Three weeks after operation the audiogram revealed that the hearing had improved 15 or more decibels in the important frequencies. In the succeeding four to six weeks the hearing continued to improve in all frequencies, finally reaching a level within 15 to 20 decibels of normal. The improvement in hearing was retained for four months. A gradual loss became evident at that time, and at the end of eight months the hearing had

dropped back to the preoperative level, and it remained there. The bone conduction was the same as it was before operation. The fistula response, which was excellent for four months, gradually diminished until it could not be elicted. This is the pattern of events usually observed when the labyrinth has not been involved by inflammation and bone regeneration has been gradual. When one is contemplating a revision, in some cases vestibular tests may afford valuable information.

TIME FOR REVISION

It is much better to wait for all inflammatory reaction to subside, so that the canal wall membrane lying over the fenestra will be extremely thin, vascularization minimal and osteogenesis completed. The best time for revision of the fenestra nov-ovalis is usually from six to twelve months after it has become evident clinically that the fenestra has closed. This rule is not absolute.

If the cartilage stopple was employed to prevent osteogenesis, the following observations may help to guide one as to the proper time for revision.

1. When a cartilage stopple is well fitted and firmly inserted into the fenestra, the result may be that there is no improvement of hearing. The reason is that one has recreated a condition which existed prior to operation, immobility of the perilymph. In such cases, in which the hearing remains practically the same, a revision may be undertaken within four to six months or earlier, depending on the appearance of the cavity.

2. When a stopple is well fitted and yet not too firmly inserted into the fenestra, and the hearing is good for several months and then slowly recedes to the preoperative level, a revision may be undertaken four to six months later.

3. Revisions may be undertaken in cases with partial improvement, never reaching the practical level of hearing, four to six months later.

It has been interesting to note at operation in some of these stopple cases that when the cartilage does not rise above the level of the surrounding bone of the labyrinthine capsule, new bone may grow completely or partially over it. When the stopple does not fit nicely in the fenestra, fibrous tissue or a little rim of bone may grow into the area between the cartilage and the fenestral margin. In cases in which the cartilage fitted the wall of the fenestra snugly, the presence of fibrous tissue or new bone was not observed. When the stopple did not extend partly into the perilymphatic space, regeneration of the endosteum was noted.

RECAPITULATION (RELEVANT QUESTIONS AND ANSWERS)

In cases that presumably were well selected for revision the findings may be so different from what one expected that prudence justifies the immediate replacement of the flap. If during reoperation it seems difficult to expose the perilymphatic space and there is fear that further

instrumentation will result in damage of the membranous labyrinth, one should stop the procedure and replace the flap over the fenestra. On elevation of the flap one may find fibrous tissue extending into the window, or the perilymphatic space may be filled with organized material. Attempted removal will generally evulse the membranous labyrinth. Leave it alone.

The importance of a history cannot be underestimated when one is considering a case for revision. Some of the relevant facts to be taken under consideration are:

1. Is this a case of malignant otosclerosis as determined from the history? In the audiogram did the air conduction curve show early loss of hearing for the high frequencies and did the bone conduction curve show a definite drop beginning with 1024 and 2048? Was there a pink reflection coming from the promontory through a translucent drum? A patient with rapidly developing otosclerosis who was operated on and whose improvement was not maintained is not a good subject for revision.

2. What was the character of the bony capsule at operation? Was the bone vascular or dense? Did it grind away easily when one was making the fenestra? Generally speaking, the more vascular the capsule the greater the probability of bone regeneration. The same will be true when the fenestra is revised.

3. Was the bony canal wall fractured during the operation, particularly that portion nearest Shrapnell's membrane? If so, a fragment may have remained attached and this may have resulted in rapid osteogenesis. In such a case if the immediate improvement of hearing was rapidly lost, reoperation is indicated early.

4. What happened postoperatively? Was the patient extremely dizzy over the usual three or four day period, or was there an interval after the first two or three days when the patient was comfortable and then, on the sixth or seventh day, there was a sudden attack of dizziness, nausea and vomiting? In patients with such a history of prolonged dizziness or late dizziness and no hearing improvement taking place, it is evident that labyrinthitis has occurred, and the fenestra should not be revised.

5. When was the improvement of hearing first noted? Usually the improvement takes place within a few weeks after the operation. In cases of mild labyrinthitis, the improvement of hearing may be delayed for weeks and then may reach the practical level. If the flap is unusually edematous, resolution of the inflammatory reaction may be slow. In such cases the improvement of hearing may be delayed. If it does come up to a high degree only to recede rather soon, one should keep in mind early fibrosis. Rapid osteogenesis may occur in the cases in which inflammation of the flap is excessive.

6. How long did the hearing improvement last before the first evidence of hearing loss was noted? Did it continue to go down slowly or rapidly? The longer the initial hearing improvement continues and the more slowly it recedes, the more suitable is the case for revision. A rapid depreciation of the improvement usually indicates fibrous invasion of the window and of the perilymphatic space.

7. Did the hearing reach the practical level in the important frequencies of 512, 1024 and 2048 at any time postoperatively? One should be reluctant to operate on a patient whose hearing did not reach the practical level at any time unless the patient was one who had a stopple inserted.

8. What did the fistula test reveal postoperatively? Was the loss of response concomitant with the loss of hearing? Has the response continued, though diminished, in spite of the hearing loss? Generally speaking, when the hearing improvement has reached a high degree, the fistula response is active, and when osteogenesis is taking place in the fenestra, the fistula response diminishes. When the window is completely closed, the response is absent. If there is the most minute opening, even though the hearing has receded to the preoperative level, there may be a fistula reaction. In patients with stopple inserts in whom the hearing improvement is lost, the fistula response may continue. When the bone has completely grown over the stopple, the response is lost. When fibrosis has obstructed the perilymphatic space, even when there is little mobility of the perilymph, the fistula response may be present. Occasionally no fistula response can be elicited and yet the hearing improvement is excellent.

COMMENT

The number of cases in which hearing is improved by revision is small. If the cases are well selected, one may expect a return to the practical level in about 20 to 25 per cent. In perhaps 15 to 20 per cent of the remainder the hearing may be improved but not to the practical level. This is a conservative estimate. The danger of the hearing being made worse by revision is much greater than at the time of the original operation.

The prevention of fibrous changes and osteogenetic activity has been, in my opinion, beyond human control. If one exercises good judgment as to when not to tinker unnecessarily at the time of revision, the chances are that one will do the patient no harm. This is my present stand in regard to that procedure.

With full realization that revisions for the most part have not restored hearing, I still believe that this procedure is justifiable. Otologists all hope that the time will come when a patient operated on for otosclerosis can expect to have his hearing improvement continue without abatement and that revisions will be unnecessary. Until that day arrives, reoperation offers a reasonable, though limited, chance of success.

MEDICAL AND SURGICAL CARE OF THE PATIENT SELECTED FOR FENESTRATION OF THE LABYRINTH

K. M. DAY, M.D.
PITTSBURGH

THAT hearing for normal speech can be restored by means of the fenestration operation is now a proved fact. Patients who have undergone this surgical procedure successfully during the past nine years are a living testimonial to this fact and are loud and positive in their praises of it. Less is heard from the growing number of people who have been operated on for the relief of their deafness without success. They have cheerfully submitted to the surgeon's care, full of hope and anticipation, some of them with the assurance that the odds were all in their favor, only to have their hopes shattered, their hearing no better and sometimes worse, frequently with an infected mastoid cavity and spells of dizziness, and usually with a much lightened pocketbook. More and more of these people are drifting into the offices of otologists, and they are indeed a pitiful spectacle.

The prospect of aggrandizement beckons, and many otologists are responding to it who are inadequately trained and incapable of handling this type of work successfully. Some form of control is badly needed if the public is to be protected and the fenestration operation saved from falling into disrepute, even to the possible extreme of a specially organized regulating board. I say this in the face of the probable accusation that a few men are trying to make a monopoly of this work. Quite the contrary is true. There is a definite need for thoroughly trained and skilled operators with a sound otologic background and a sympathetic understanding of deafness and its problems. Facilities for providing such training must be made available. Too many men consider the fenestration of the labyrinth as just another surgical procedure which can be mastered by a few weeks' training on the cadaver. This operation is quite different from most of the operations performed on the ears, the nose or the throat, from the effects of which many patients recover in spite of indifferent surgical skill. The technical performance of this operation must be exact. Great accuracy and precision are required and much of the procedure is microscopic. It is not an

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operation to be performed by a surgeon with heavy hands, poor vision or a lack of thorough understanding of fundamental surgical principles.

There are three primary causes of failure with the fenestration operation: 1. Faulty diagnosis and selection of cases in which perfectly performed operations will not restore practical hearing. 2. Fibrosis occurring in the vestibule from hemorrhage, entrance of foreign material or damage of the vestibular contents. 3. Closure of the fenestra.

It is my contention that closure of the fenestra is due not merely to the natural tendency of bone to regenerate but to bone dust, endosteal tags or spicules of bone left in the fenestra or on the flap covering it, or else to inflammatory reaction which provides the stimulus needed to cause bony regeneration. In other words, the faults which cause bony closure lie, I believe, with the operator; a clean fenestra, covered by a clean and healthy flap, will not close in the absence of the stimulative inflammatory reaction.

Every surgeon who undertakes this work should keep careful records of his cases, recording all minor technical faults and mishaps which occur during the operation and postoperative dressings. If a closure of the fenestra results in any given case and the operator has a good surgical conscience, he usually will be able to detect the faults or mishaps which contributed to that closure. Then he will be better prepared to avoid making similar mistakes in the future. It is a grave offense to shrug aside one's failures as due to an act of nature or of God.

Aside from the technical procedure of the operation itself there are many minor details associated with the medical and surgical care of the patient which undoubtedly influence the well-being of the patient and the eventual outcome of the operation.

It is presumed that only people who are in good physical condition and who are good surgical risks are accepted for operation. In the medical examination before operation, special attention should be paid to the possibility of active nasal allergy. I consider that in my series of cases acute nasal allergy occurring within two months after operation was at least partially responsible for four failures Two of these failures concerned patients in whom a secretory condition of the middle ears developed. The other two occurred in patients whose ear drums were distended and meatal flaps reddened as a result of sneezing and blowing.

Psychologic instability is often associated with physical handicaps, and I believe this is especially true of the hard of hearing. It should be realized that the psychic shock from having one's hearing restored may be just as severe as the shock of failure. In 2 patients I observed active manifestations of dementia precox develop after successful fenestration. This fact should be considered during the preoperative medical

examination, and any patient whose stability is in doubt should be referred to a psychiatrist.

If local anesthesia is to be used for the operation, an explanation should be given to the patient of what he can expect to experience during and after the operation. I rarely have difficulty in controlling my patients with local anesthesia if they know beforehand about the loud, jarring noises and spells of vertigo which occur during the operation. Otherwise, some patients translate the sensation of noise into actual pain. If one of the barbiturates is to be injected intravenously for anesthesia or for sedation in association with local anesthesia, it is advisable to give a mild dose the night before operation in order to be sure that the patient is not allergic to barbiturates. An occasional catastrophe may be avoided by this precaution.

Liberal ingestion of fluids is encouraged up to within two hours of operation. If the operation is scheduled for early morning, it is wise to have the patient wakened, if necessary, two hours earlier and given a glass of fruit juice. Fluids are also given freely by mouth after operation. It is unusual for a patient to vomit more than once or twice after operation if the ingestion of fluids is unrestricted, and I rarely find the administration of surgical fluids to be necessary or desirable.

During the surgical preparation of the operative field, it is well to prevent mercurial or iodine solutions from entering the external meatus. Occasionally the skin may prove to be sensitive to such solutions, as shown by a marked inflammatory reaction. This is most undesirable, and an operative failure might result if the tympanomeatal flap should undergo such a reaction. Filling the external meatus with 70 per cent alcohol for ten minutes and then closing it with sterile cotton has proved to be quite adequate for antisepsis, and I have seen no operative infections after employing this procedure.

Some comment should be made about the packing used in the cavity at the conclusion of the operation. The packing should be nonirritative, nonadhesive and nonobstructive. Paraffin gauze is satisfactory in these respects. Pieces of cellulose sponge can be molded nicely into the cavity and have the advantage of their ability to absorb excess secretions, but they have a tendency to adhere to the walls of the cavity and may produce bleeding on removal. There seems to be a general tendency to insert too much packing and to pack too tightly in order to insure that the tympanomeatal flap will remain in position. This may be harmful, as tight packing may choke the flap and partly cut off its circulation. It also causes pain. Pain means inflammatory reaction, and inflammatory reaction provides the stimulus for regeneration of bone. If the operative wound is properly cared for, there should be little if any pain associated with this operation. Gentleness should be a watchword in surgery and should apply equally to the handling of tissues during

operation and to the care of the wound afterward. Packing should be inserted gently and removed the same way. Great care should be exercised in the removal, so that the packing is lifted away and not rubbed along the surface of the flap. It is during this procedure that a secondary hemorrhage might occur beneath the flap, and this might result in an operative failure. I have found that all packing can be removed from the cavity by the fifth postoperative day, by which time the flap is securely sealed in position. After the packing has been removed, the less the cavity is meddled with the better the patient seems to get along. For the next few weeks local treatment usually is limited to cleansing the external meatus and mopping out excessive secretions when necessary.

Lately, it has been my practice to encourage my patients to sit up on the day of operation, with use of a backrest, and to get out of bed on the day following. Within twenty-four hours after operation most patients are able to use the bathroom and to sit in a chair while the bed linen is changed, without excessive vertigo or labyrinthine disturbance. It is surprising how quickly the labyrinth becomes adjusted, and vertigo subsides when this procedure has been followed. Normal stimulation of the vestibular mechanism seems to hasten the adjustment rather than to retard it. Moreover, audiograms of these patients taken at the end of three or four weeks definitely show less reduction of hearing for the higher frequencies than was observed when patients were confined to bed for several days after operation. Many of my patients are now walking around on the second and third postoperative days and are experiencing little or no vertigo. There is an occasional exception when the patient must be confined to bed for several days because of an active labyrinthine disturbance, but this is an exception and not the rule.

The postoperative care of the cavity is often a real problem. The healing properties of wounds resulting from the same type of operation vary tremendously. In some cases the cavity heals promptly, in a few weeks, without showing any evidence of infection in spite of lack of care and cleanliness on the part of the patient. In others, secondary infection of the cavity may occur early in spite of strictest precautions and persist for weeks or months. In still a third group of cases, excessive granulations may form and require repeated removal before the cavity heals. Too many patients are discharged as well before healing is completed and acquire secondary infections which persist indefinitely. Both the operation itself and the after-care of the cavity are individualized problems which often tax the surgeon's knowledge, judgment and ingenuity.

Some of the comments I have just made about medical and surgical care may seem rather elementary and of minor importance. It might

be well for all to remember the old adage attributed to Michelangelo that trifles make perfection, but perfection is no trifle.

Continued research to determine factors which promote and retard osteogenesis is certainly desirable; also methods of simplifying and perfecting the present operative procedure. In the meantime, I believe that improved results can be obtained, as well as a higher percentage of successes, by constantly striving to improve one's technical performance and by paying closer attention to apparently minor details.

121 University Place.

TRAINING OF SURGEON AND SELECTION OF PATIENT FOR THE FENESTRATION OPERATION

J. H. MAXWELL, M.D.
Professor of Otolaryngology, University of Michigan Medical School
ANN ARBOR, MICH.

THE TRAINING OF THE SURGEON

THE TRAINING of the surgeon and the selection of the patient are two inseparable and eminently important problems in the overall aspect of fenestration surgery. A surgeon who is poorly trained in this field cannot select properly the candidate who is suitable for the Lempert fenestra nov-ovalis operation. A surgeon trained only in the technic of this operation is not fully equipped for surgical treatment of patients with otosclerosis. If he has any inclination to adapt patients to a form of therapy in which he is desirous of gaining a rich experience, his selection of patients will be unfortunate.

Apparently, the technical training of most surgeons performing the Lempert operation has been fairly adequate from the standpoint of anatomic dissection. It should not be forgotten, however, that the operation is something more than an anatomic dissection to be performed on the living patient. It is a major surgical operation involving vital structures and requiring for its satisfactory performance a high degree of technical skill. The prerequisite training of the otosclerosis surgeon should include first of all a wide background of diagnostic and surgical experience in the general field of otology. After he has studied the entire problem of deafness in all its phases, this well disciplined otologist is ready to prepare himself to perform the fenestration operation. In the final preparation it is generally advisable for him to go to an institution such as Lempert's laboratory where he can do the operation on cadavers under supervision, and also have many opportunities to observe experienced surgeons perform the operation. It is most important for the beginner to study the skills by which experienced surgeons surmount the various technical difficulties of the procedure.

The fact that there is a steadily increasing number of men who are equipping themselves to perform the fenestration operation is both

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encouraging and distressing. Not all of these men are associated with large clinics or even practice in large metropolitan areas. Many of them are young men who have had barely enough training and experience to pass their American Board examinations. Their desire to progress and acquaint themselves with recent advances in otologic surgery is admirable but their eagerness to perform the fenestration operation may be cause for some concern.

Since 1938, when Dr. Lempert first reported his one stage technic for the fenestration operation for clinical otosclerosis, several thousands of patients have undergone this operation. For the most part, these patients have been treated by skilful surgeons of wide experience who have made every effort to select cases in which they had a justifiable right to expect good results and who have followed their cases with genuine scientific interest. Since 1938, or more logically since 1942, when the number of the patients operated on increased sharply, a large percentage of patients of all ages who might be candidates for this surgical procedure have been examined and have either undergone the operation or have decided on other means of rehabilitation. From now on most of the surgical patients will be recruited from the younger persons who are beginning to be handicapped by the impairment of their hearing. Therefore, it would seem obvious that a diminishing number of candidates for the fenestration operation might be anticipated. This fact assumes importance only when one considers the large number of enthusiastic otologic surgeons who are becoming technically equipped to perform the operation. That economic pressure and the desire for a wide surgical experience may become factors in poor selection of surgical cases can scarcely be doubted. It is therefore my earnest plea that otologists who desire technical training in otosclerosis surgery be scrutinized most carefully and chosen by those who supply facilities for their training with the same care that is used in the selection of residents and instructors in the larger teaching hospitals of this country. If it can be made practicable, it would be well for the national societies to elect a rotating board of otologists to determine the qualifications of otosclerosis surgeons and to issue certificates of proficiency in this field of surgery to applicants who meet prescribed requirements.

THE SELECTION OF THE PATIENT

It is readily admitted that there is considerable latitude in the selection of patients suitable for the fenestration operation. Any attempt to draft hard and fast rules fixing the degree of handicap, the exact positions of the air conduction and bone conduction curves on the audiograms or the age limits in regard to this operation is entirely beyond my province and capability. Certain principles, however, must be held inviolable,

and after careful attention has been given them, the final decision must be based on clinical experience moderated by common sense.

In the first place it is obviously necessary to make a reasonably accurate diagnosis of the hearing impairment of which the patient complains. The conductive deafness which may lead to a diagnosis of clinical otosclerosis must not be determined from the audiogram alone or from the audiogram in addition to a casual and superficial objective examination. Nasopharyngeal examination and otoscopy to exclude tubotympanitis or adhesive otitis as the cause of the patient's impairment of hearing are time consuming but are important parts of the examination. The significant features of the history of a patient suffering from clinical otosclerosis are well known to all otologists. However, the rapidity with which the impairment of hearing has developed to the level of a social handicap and the length of time during which there has been no noticeable progress of the deafness are features which too many times have been considered lightly by the examining otologist. If a patient over 50 years of age has an impairment that has not progressed noticeably during the ten years, say, prior to his examination and if he has maintained his social and economic position with or without the assistance of a hearing aid, the fenestration operation is rarely indicated even if careful studies denote the presence of clinical otosclerosis. The young person who complains of deafness that has developed and progressed to the degree of a real handicap within a few months should arouse some anxiety in the mind of the examiner. Rapidly developing clinical otosclerosis has a propensity for continuing on its deleterious course even after a well performed fenestration.

The audiogram is the result of a subjective test, and as such it records the vagaries of the patient responding to sensory stimuli. It is highly desirable to have a series of three or four audiograms taken several days apart in an effort to gain accurate information regarding the acuity of the patient's hearing of pure tones. The hearing should then be tested with tuning forks. Although I agree with those who maintain that bone conduction tests alone are not thoroughly reliable indicators of cochlear function, they should be used routinely since they give valuable information in the vast majority of cases. The candidate for fenestration should have a strongly negative Rinne test and a positive Schwabach test with the C 3 (1024 double vibrations per second) tuning fork, and with few exceptions he should lateralize sound in the Weber test to the worse ear when the C 3 and C 4 forks are used. I have found no thoroughly satisfactory means of testing bone conduction with the C 4 (2048 double vibrations per second) tuning fork on each ear separately. However, if both external canals are closed by pressing the tragus firmly into the meatus on each side, the Weber test may be done with the C 4 fork. If in the Weber test sound is thus lateralized to the

worse ear or is definitely heard on both sides, it is a good indication of the patient's ability to hear the C 4 fork by bone conduction. The chief value of the tuning fork test is to check the audiogram, which on many occasions is found to be inaccurate in spite of frequent calibration of the audiometer. Also, the tuning fork tests, if carefully done, may permit one to make a decision regarding the advisability of fenestration in a borderline case. Comparing the results of the Rinne test obtained by using the C 2 (512 double vibrations per second) fork and the C 3 (1024 double vibrations per second) fork and testing the patient's ability to hear the C 4 (2048 double vibrations per second) fork by bone conduction are helpful in this regard.

In addition to the tuning fork tests and pure tone audiometry, the testing of speech reception is most important, for it is obvious that the patient's greatest interest is in his ability to hear speech. It is to be hoped that the time will soon come when all reported results of the fenestration operation will show the preoperative and postoperative thresholds of speech reception. The degree of the patient's handicap and the success of the operation can be shown to much better advantage by this means. The present tendency to conclude that a patient is handicapped or that the fenestration has not been successful if there is slightly more than a 30 decibel loss for just one or even two of the tones in the critical frequencies as recorded on the audiogram seems to me to be absurd. If a patient handicapped by impairment of hearing has been rehabilitated by fenestration so that he can carry on normal group conversation, the operation has obviously produced a satisfactory result. True, the patient will show on an audiogram hearing losses of less than 30 decibels for the tones of the critical frequencies in most instances, but in some instances there may be a loss of 40 decibels for one or possibly two of these tones. Considering the limit of error in the audiogram and the patient's apparent rehabilitation, the latter group should be considered to show successful results from the operation. These instances certainly would be classified among those in which good results were obtained if reports were based on speech reception tests.

I believe that no patient is suitable for the fenestration operation unless it can be shown by accepted tests that there is sufficient cochlear function to permit the hearing to be restored to a serviceable and practicable level through the successful by-passing of the obstructive element. It is true that some cases have been reported in which serviceable hearing has been restored by fenestration although the audiograms indicated profound nerve deafness. These cases are rare and offer no justification to the otologist for operating on patients with evidence of extensive cochlear damage in the hope that there may be some improvement in hearing even though it will probably be below the serviceable level.

Handicapped persons will grasp at straws. The patient with profound deafness is willing to try almost anything that offers even a faint ray of hope that his affliction will be ameliorated. Many times such a patient will beg the surgeon to do the fenestration operation even though he has been told that he is unsuitable. The performance of the fenestration operation as a surgical experiment is a violation of the ethical concepts of the profession. As a surgeon, one does not have surgical commodities to sell! One has opinions and advice to offer. If a surgeon feels that the operation should be done, he should so advise the patient. If, according to available and accepted hearing tests a patient is classified as unsuitable for the fenestration operation, the operation should not be recommended. This patient should be apprised of his condition and given advice and assistance in regard to rehabilitation through speech reading and the use of a hearing aid.

GENERAL CORRELATION

MARVIN F. JONES, M.D.
NEW YORK

THE PAPERS presented have stated their message in a clear simple, understandable manner. I will endeavor to follow their example in my comments.

The value to be derived from a symposium such as that presented in the foregoing pages lies in a frank discussion of authoritative opinions. All the authors have based their opinions on their experience. The main problem is to reduce failures. This problem, in my opinion, has not been solved. The entire paper of Dr. Lempert impressed me as an honest statement of his efforts and hopes to perfect his own nov-ovalis endaural fenestration operation. Osteogenic closure of the artificial fenestration still remains a major cause of failure. The various procedures for preventing closure of the fenestra which have been tried and discarded are mentioned. Two methods employed by Lempert to prevent closure still hold some promise of success. These two methods may still be considered as in the experimental stage. Use of the lead burr appears to be a method of preventing closure, but lead may have other deleterious effects. Lead is not an inert substance, and lead particles remain after the burr is used. It should not be recommended for general use at this time.

The cartilage stopple is another promising agent used to prevent closure. In his paper Lempert appears hopeful that proper use of the stopple may finally solve the problem of closure. However, in other hands the use of the stopple has not produced results which would satisfy the high expectations raised by former enthusiasm. Various experiments which have been and are now being conducted by Lempert indicate that the stopple has not been entirely satisfactory even in his hands.

Lempert uses the word "predict" in relation to the final result of surgical treatment. This word is aptly used. The power to predict lies in the ability to make a correct diagnosis and in knowing which fenestrations will close. He mentions the inadequacy of the present diagnostic tests and the consequent inability of the physician to predict results of surgical treatment when indications are based on these tests. I am glad

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to see that Lempert has already embarked on research in this potentially useful diagnostic field.

One factor which Lempert mentioned as contributing to bony regrowth is bleeding. He asserts that he does not place the flap over the fenestration until the field is free from all blood or bleeding. I have been unable to obtain a blood-free field consistently with any type of general anesthesia. I know that the use of an anesthetic such as Lempert and Branower advocate does permit the operator to obtain a blood-free closure. My own experience makes me believe that blood encourages and aids bony regrowth.

In the hands of Lempert and Branower their method of inducing anesthesia may be considered safe. I have not shared their feeling of safety in regard to the method. Time must develop a safe anesthesia with a blood-free field to insure the best results.

A moratorium should be called on the flaunting of percentages, and I am using the word "flaunt" in the sense Webster permits, i.e., "display boastfully." The percentage reported by Lempert is not high enough to permit one to be boastful. There are so many variable personal and mechanical factors which may affect these reports that even a discussion of these factors would lead to an interminable argument. I am sure many of the discrepancies are known to all. When percentages which have been published range from 38 to 95 for surgeons of equal ability, it is time for physicians to scratch their heads and wonder.

The present standard for reporting is unsatisfactory, but it is the best of any so far proposed. This standard is a threshold of hearing above the 30 decibel level of loss for the three frequencies in the conversational range (512, 1024, 2048). It would be most desirable to have some standard of reporting approved by the American Otological Society.

Lempert has included the results of animal experimentation in his paper. According to my understanding, this work was a joint effort participated in by Lempert, Schall, Meltzer and Dorothy Wolff. Since there is another paper on their work included in the present symposium I shall not comment further at this point.

As a final note on Lempert's paper I should say that the many discouraging trials and some encouraging trials of methods to prevent bony closure offer sufficient evidence for the conclusion that no method used thus far insures permanent patency of the surgical fenestration. Some are effective, but none is consistently reliable.

The second paper, presented by Dr. Schall, with Lempert, Meltzer and and Dorothy Wolff as co-authors, is an authoritative statement of experimental observations as interpreted by themselves. Knowing the ability of the authors, I would not question their deductions. Their report of

the research which was done on the rhesus monkey can be accepted as true for the rhesus monkey. Its application to the human subject has been extended by Lempert. Their conclusions seem logical and correct. It can be accepted that bone regeneration of the labyrinth can be controlled by the lead burr when this is used on the rhesus monkey. This brief statement does not express adequately the value of a carefully controlled research which resulted in their important conclusions.

Dr. Day has fortunately digressed from his assigned subject, "Medical and Surgical Care." In this digression he accentuates one of the two main themes of Dr. Maxwell's paper. He refers to the training of otologic surgeons in the fenestration technic. The fact that these two authors have called attention to the same point assures me that premonitions expressed in my report to the American Academy of Ophthalmology and Oto-Laryngology were well founded. I will not add useless repetition to the plainly expressed statements, but I should like to sanction remedial measures. The persons or institutions engaged in the special training for fenestration surgery should be authorized by some official body. The physicians who engage in fenestration surgery should be certified by the American Board of Otolaryngology. I hope some action will be taken immediately to initiate such control. Day believes that the faults which cause bony closure lie with the operator and that a clean fenestra covered with a clean, healthy flap will not close in the absence of inflammatory reaction. This, as stated, is his belief but were this literally true many surgeons now performing the fenestration operation would have had no closures. I can hardly believe that Day meant his statement to be quite so dogmatic as it sounds.

He wisely recommends gentleness in this operation. Careful manipulation of tissues has been recognized by the general surgeon as a major factor in accelerating wound healing. It is most commendable to have an otologic surgeon stress this most important point. The fenestration operation, above all others, demands a deliberate delicacy and gentleness in handling tissue. Day also stresses the same theme for postoperative care. One who has been on the receiving end of surgical treatment appreciates the presence of a "light touch" in his surgeon's hands.

Day takes another leaf from the more recent surgical annals and recommends a short confinement to bed. I find much less tendency toward vestibular disturbances and their consequences when the patient remains prone and quiet.

Day is so right when he stresses perfection in apparently minor details. This work demands precision, and this precision must be retained from the patient's first visit to the final discharge. Every detail is important.

Dr. Maxwell has been assigned two of the three urgent problems requiring action. They are the training of the surgeon and the selection of the patient. A third which I would include is an officially accepted standard for reporting results. With one exception Maxwell's paper states his message in an understandable and understanding manner. This one statement seems so out of place in such scholarly surroundings that it makes me wonder whether I fully grasped his meaning. He states: "The performance of the fenestration operation as a surgical experiment is a violation of the ethical concepts of the profession." I hope Maxwell will consider the implications of such a statement as applied to all innovations in medicine and especially as applied to fenestration surgery. I would suggest deletion of this statement from his published paper if, after conscientious thought, he feels it advisable.

As Maxwell has said, "More attention should be paid to the education in diagnosis preliminary to technical training." During a recent Board examination I asked candidates to describe the fork tests used in making a diagnosis of otosclerosis. Only one candidate mentioned any fork of higher frequency than 256 double vibrations per second. Selection of patients who could be benefited by surgical treatment would be impossible if the physician possessed such inadequate knowledge.

In his paper Maxwell has presented his subjects in such a way that they should serve as a text for future guidance. Any further comment would be "gilding the lily."

Dr. Meltzer has presented his usual thoughtful, modest and honest paper. The mere fact that the subject of revision is a part of this program is proof in itself that the problem of closure is far from an acceptable solution. He states that "ultimate failures are of such frequency that the question of revision becomes one of major importance." This statement shows that in his mind the problem of the bony closure is still to be solved. In Meltzer's paper I detected a nuance of expression which carries force because of its unassuming subtilty. His eight questions and answers could have been presented only by an observant, experienced and capable surgeon. His answers may well serve as a guide for those surgeons, including myself, who still get bony closures. Such an honest statement as "The number of cases improved by revision is small" appeals to me because it has been my own experience.

We are in agreement also regarding the greater danger to hearing incurred in the revision. The question naturally arises regarding the advisibility of secondary operations. Even if the hearing of 1 out of 5 is permanently improved, I agree again with Meltzer, it seems worth the risk. His final statement should be engraved on all papers dealing with fenestration: "This is my present stand in regard to this procedure."

LESSONS TO BE LEARNED FROM THIS SYMPOSIUM

1. There is a definite and too great element of error in the present methods of arriving at a diagnosis which precludes dogmatism.

2. Training methods for the development of understanding, capable fenestration surgeons should be subject to the approval of authoritative bodies.

3. Certification of those trained in fenestration surgery should be implemented by the American Board of Otolaryngology at once as a protection to the public.

4. Anesthesia for fenestration should receive more attention on the basis of blood control and safety.

5. The Lempert endaural fenestration operation performed by a competent otologic surgeon, properly trained, improves hearing to the useful level. The patient's hearing can be maintained for ten years in an indefinite percentage of selected cases. The figures reported thus far are so divergent that it seems inadvisable to state a definite percentage. I would feel justified in saying there is a permanent improvement in more than 50 per cent.

A comment which I deem in order concerns the medical information liberated to the lay press. The public is entitled to know the applicable advances in medicine. Unfortunately, there has been no distinction between reporting experimental progress and reporting established procedures. The first enthusiastic statements are not apt to be the final, soberly considered deductions. The old pendulum idea holds also in medicine. The first flush of optimistic endeavor is sometimes followed by an overly pessimistic phase as the other extreme. Opinion will finally settle down to correct values. This correlation and evaluation of the symposium is, as Dr. Meltzer says, "my present stand."

121 East Sixtieth Street.

Case Reports

SUCCESSFUL AURICULAR AUTOTRANSPLANTATION

ALFRED WACHSBERGER, M.D.
SACRAMENTO, CALIF.

AUTOTRANSPLANTATION of an organ, whole or part, is an experimental surgical procedure. It is usually unsuccessful; although the literature contains rare reports of a take, it generally discourages attempts of that sort.

REPORT OF A CASE

A case in which a part of one auricle was successfully transplanted to the other is reported herein for the sake of recording such an oddity.

G. A., a 41 year old man, received an accidental injury to his left ear twelve years ago, which resulted in a defect of the auricle. He asked for repair of this deformity because of the stigma and because of the ridicule by his fellow workers.

Examination gave essentially negative results except for a hernia and the deformed left auricle. There was a triangular defect in the posterior half of the concha measuring 12 by 15 by 40 mm., involving the helix, the anthelix and the cymba conchae.

The following observations were considered in planning the plastic repair of the deformity: Both ears were proportionately large. The best cosmetic result could be obtained by freshening the edges of the defect and by adaptation, with secondary excision of a full thickness wedge—Joseph technic for the general reduction of an oversized auricle—if necessary. This, of course, would reduce the size of the left ear, which in itself would be of proportionately good size, but much smaller than its normal mate. The right ear would then have to be made smaller in order to obtain two ears of equal size. Because the right ear had to be reduced in size anyway, it was decided to attempt to graft the primary wedge, excised from the right ear, and transplant it to the freshened defect of the left ear. This was done on April 15, 1943. Secondary wedge excision was necessary for coaptation of the right ear, but not for the left ear. Fine silk sutures were used and were led through the skin only, not through the cartilage. Postoperatively, a hot water bag was kept near the left ear, over the bandage, for eight days. The dressing was changed every three days, and the sutures were removed at the time of the second dressing. Healing was uneventful and resulted in a perfect take about May 3, or eighteen days after the operation. The skin of the graft presented normal color and texture on the concave as well as on the convex side. Minor irregularities of the wound edges were corrected on May 12, and this resulted in a smoothly sculptured ear. Both ears were equal in size.

1025 Ninth Street.

Fig. 1.—*A* and *B*, preoperative and postoperative front view of the left ear. *C* and *D*, preoperative and postoperative back view of the left ear.

Fig. 2.—Appearance of the corrected ears.

Abstracts from Current Literature

Ear

THE DEVELOPMENT OF RECORDED AUDITORY TESTS FOR MEASURING HEARING LOSS FOR SPEECH. C. V. HUDGINS, J. E. HAWKINS, J. E. KARLIN and S. S. STEVENS, Laryngoscope **57**:57 (Jan.) 1947.

Two auditory tests, identical in design, one using words and one short sentences, are described. Both measure the hearing loss by a comparison of the threshold of hearing of the normal person with that of the person with impaired hearing. This difference must be known in decibels and under identical conditions. The absolute value of the speech intensities need not be known.

The essential characteristics of the test materials are discussed in detail. Spondees (i. e., words such as "blackbird," in which both syllables are equally accented) were found to be the best. Lists of words and of sentences are given. The equipment needed and the method of use are described. Under such circumstances the tests were found to be reliable. A speech test by which high tone loss can be differentiated from a flat loss of hearing was devised. It showed no real advantage over the pure tone audiometer for this purpose.

HITSCHLER, Philadelphia.

SYMPOSIUM ON NOISE: THE PERCENTAGE OF CAPACITY TO HEAR SPEECH, AND RELATED DISABILITIES. EDMUND PRINCE FOWLER, Laryngoscope **57**:103 (Feb.) 1947.

Fowler discusses the pure tone audiogram in relation to the capacity to hear speech. The percentage loss of hearing as determined by means of the American Medical Association's chart with weighted figures sometimes is at great variance with the person's ability to hear speech. One of the main difficulties of this method of computation is that a constant factor (7) is used in determining the relative values of the hearing in the better and the poorer ear. The factor should vary according to the amount and the difference of hearing between the ears. There are several means of calculation which will give more accurate results, the simplest being briefly as follows: Multiply the percentage loss of the better ear by 10, and that of the poorer ear by one tenth of the loss of the better ear. Add the two results and divide by the sum of the multipliers.

No mechanical means, however, will take into account the person's own ability to get along with a hearing defect. Some with large defects apparently have a greater capacity to hear speech than those with lesser defects.

HITSCHLER, Philadelphia.

ANATOMICOCLINICAL CLASSIFICATION OF THE LABYRINTHINOPATHIES. MARIO RIUS, An. de oto-rino-laring. **16**:51, 1946.

Rius has attempted to classify the various alterations of the labyrinth from both an anatomic and a clinical standpoint. His extensive monograph includes case histories, audiograms, vestibular findings, methods of treatment and the results. He classifies the alterations as follows:

Group 1. Congenital malformations, such as aplasia and atresia of the auditory system. The dominant clinical finding is deaf-mutism.

Group 2. Lesion of the labyrinthine capsule. Either there is a reaction to the fistula test or there is generalized alteration of the capsule. The former is associated with a suppurative condition of the mastoid process, syphilis or fracture of the capsule. The latter is caused by infection, trauma, various metabolic, endocrine and bony dystrophies and otosclerosis.

Group 3. Infectious labyrinthitis. This may be either pyogenic or nonpyogenic.

Group 4. Proliferation of the connective tissue stroma of the internal ear, producing obliterative otitis interna, associated with fracture, suppurative otitis media, suppurative meningitis or syphilis.

Group 5. Vascular changes, either arterial or venous, or blood changes, producing many of the symptoms complained of by the older persons and associated with such conditions as menopause, shock, cardiac insufficiency, tumor of the acoustic nerve, syphilis, arteriosclerosis, thrombosis or embolism of the internal auditory artery.

Group 6. Alterations of the dynamics of the endolymph as evidenced by hydrops of the labyrinth as in Ménière's disease; acute changes of air pressure as in aviation; periendolymphatic hypertension as seen in congestive internal otitis or in arachnoiditis.

Group 7. Degenerative conditions of the labyrinth, particularly of the neuroepithelium, which may be primary or idiopathic (e. g., congenital deafness, hereditary deafness and premature presbyacusia) or secondary to trauma, operation, tumor of the acoustic nerve or toxic influences (from suppuration or such a condition as diabetes or uremia, or overdosage of quinine or salicylates or poisoning with arsenic, lead, phosphorus, mercury or the aniline dyes) or to avitaminosis.

Group 8. Labyrinthine disturbances of a reflex nature from a central or a cervical region, such as trophoneurotic lesions of the bulb or lesions of the fifth nerve and its branches.

A detailed and comprehensive description is given of the causes, the symptoms and the treatment of each of the various conditions.

In this article Rius has made an extremely valuable conribution to present knowledge of the pathologic conditions of the inner ear. It requires a considerable amount of careful study, but it is of great value, particularly from the clinical and the therapeutic standpoint, and the author should be complimented on his excellent work.

PERSKY, Philadelphia.

TRANSITORY DEAFNESS. REIDAR MOE, Acta oto-laryng. **35**:20 (Jan.-April) 1947.

Deafness presupposes a lesion of the sound-perceptive mechanism, that is, the peripheral end organ or the pathway by which sound stimuli are transmitted to the brain center. The pathway, in brief, is as follows: The central fibers of the bipolar cells of the spinal ganglion assemble to form the cochlear nerve which travels through the internal auditory meatus to the ventral and dorsal cochlear nuclei in the medulla. From here some of the fibers of the second neuron travel centrally on the same side, but most of them travel by three separate bundles to the opposite side, to unite with the noncrossing pathways from the other ear, and pass through the lateral lemniscus to the posterior corpora quadrigemina to the medial corpus geniculum. From here the pathways go to the center in the temporal lobe, which lies on the inner side of the upper temporal convolution in the fossa of Sylvius. Thus the majority of the central cochlear pathways cross. A single focus can therefore affect all of the cochlear fibers from one ear either in the acoustic nerve itself or immediately after these fibers enter into the medulla. If all the fibers

are to be attacked centrally there must either be several foci or one that is large enough to affect the central tracts of both sides. A lesion of the lateral lemniscus can effect considerable loss of function on the opposite side and lesser impairment on the same side.

By far the most common site of pathologic processes leading to deafness is in the labyrinth. It is usually difficult to decide whether the lesion is in the labyrinth or in the cochlear nerve, but Ruttin believes that when vestibular symptoms are present at the same time, the primary lesion is in the labyrinth, while the absence of these vestibular symptoms speaks for a location in the cochlear stem.

Disturbances of the sound perceptive mechanism may arise suddenly, sometimes in apoplectic form, or they may develop slowly. Usually their course is either progressive or stationary and is not subject to great fluctuations except in the purely vasomotor derangements, such as Kobrak's "octavus crises," Pals's "vascular crises" or Brunner's "otitis interna vasomotoria." The prognosis is, on the whole, unfavorable. Klestadt says that permanent restoration of hearing may be noted even in the case of the greatest defects both in peripheral and in central lesions, but Moe says that permanent restoration in cases of complete deafness is exceedingly rare.

Some patients with serous labyrinthitis recover their hearing. In many cases of infectious toxic neuritis of the acoustic nerve with complete loss of function, some recovery of function may take place. Recovery of hearing has been reported in a number of cases of loss caused by the virus of herpes zoster oticus. Moe has found record of only 1 case in which mumps had caused loss of hearing of a transitory nature. Cases of transitory deafness occurring in malaria and in yellow fever have been reported by Kravcenko and by Alexander, respectively. Both Grahe and Alexander state that the acoustic damage caused by encephalitis lethargica presents a variable course. Moe found 3 cases of transitory deafness following vaccination or serum treatment cited in the literature. Numerous cases of transitory deafness associated with syphilis occur. In some of these the deafness is undoubtedly due to the syphilis itself and in some it may be due to the treatment with arsenicals. Transitory deafness has been observed in cases of poisoning with arsenic, silver nitrate, atoxyl, arsphenamine, illuminating gas and oil of chenopodium.

Disturbances of hearing and especially complete cessation of function of the cochlear nerve are rare in cases of organic disease of the brain, but transitory deafness is encountered during the course of multiple sclerosis.

Transitory disturbances of hearing are also found in cases of tumor of the brain, in which the hearing improves after the cause is removed. The exception to this statement is found in cases of tumor of the cochlear nerve or of angle tumor.

In trauma of the head the acoustic nerve is often involved even after apparently minor degrees of injury. The deafness occurring after fracture of the labyrinth is always permanent. Another important form of deafness occurring after injuries of the head is a psychogenic deafness, which is transitory. Otologists often see a combination of psychic and mechanical trauma in persons in whose vicinity there have occurred explosions or violent noise. There are, however, other forms of organic trauma in which deafness may be of a transitory nature. In these it may be due to bleeding into the labyrinth or into the nuclei.

After this exhaustive survey of the literature Moe cites 3 cases of his own.

Case 1.—A man had had bilateral otitis media for years and had had a dead labyrinth on the left side for ten years. He had some pain in the right ear, and a polyp was removed. On the following day he had complete facial paralysis. At operation a small cholesteatoma was found, but no fistula in the facial canal. A few days later he became totally deaf in the ear operated on but showed no signs

of vestibular disturbance. In the course of some months the facial paralysis cleared up and the hearing in the surgically treated ear returned to its preoperative level. Inasmuch as total deafness following a radical operation, whether it be due to labyrinthitis or to postoperative degeneration in the sense of Alexander and Brown, invariably has a bad prognosis as to return of function, Moe believed that in this particular case he was dealing with coexisting chronic otitis and herpes zoster. No herpetiform lesions were found, but he states that herpes zoster without herpes can occur. In polyneuritis due to the virus of herpes the facial nerve is often paralyzed first, as in his case, and the acoustic nerve somewhat later.

CASE 2.—A man aged 22 entered the neurologic division of the hospital with an involvement of the pyramidal tract from multiple foci. At first the hearing of the left ear was affected by a conductive lesion, which was relieved by inflation of the eustachian tube, while the hearing of the right ear was normal. Some months later he became much worse, with vertigo and nystagmus so severe that he had to remain in bed. Shortly thereafter he had severe transitory paralysis of the right leg and somewhat later incontinence. Three weeks after the appearance of the paralysis of the leg he suddenly became deaf in the right ear, which had previously been found normal. This deafness just as suddenly disappeared after it had been present for three weeks and the hearing has since remained normal. During the period in which the right cochlear nerve had ceased to function he was so dizzy that he would not permit a vestibular examination.

The diagnosis from the department of neurology was that he was suffering either from multiple sclerosis or from acute disseminated encephalitis. A differential diagnosis of these two conditions is often difficult. The fact that the condition remained stationary for many years after recovery rather points to the second of the two. Transitory deafness is occasionally seen in multiple sclerosis but only rarely. Paralysis of cerebral nerves, with the exception of the optic nerve, is also quite rare in encephalitis. The disease probably attacked the cochlear nerve immediately after its involvement of the medulla.

CASE 3.—In a child of 12 months, previously healthy, signs of an acute cerebral disease, with convulsions, developed after tonsillitis. After she was hospitalized mild acute bilateral otitis media developed. The attacks of convulsions gradually ceased, and she was sent home. Shortly thereafter mumps developed, but before this appeared, it was noted that she was completely deaf in both ears. At this time a caloric test of both ears gave a negative result. Her hearing gradually improved. In the following year she again had convulsions and again became deaf, but on this occasion the vestibular apparatus gave normal reactions. By degrees the hearing returned and has remained normal in spite of fresh convulsive seizures. The diagnosis of postinfectious encephalitis was made.

As an etiologic factor for her deafness the following conditions must be considered: (1) the acute otitis media, (2) the recent parotitis and (3) the encephalitis. Even mild acute otitis media can produce labyrinthitis, but it would be rare for it to do so, and it would be still more rare for it to attack both labyrinths simultaneously. However, the child did not present any clinical signs of labyrinthitis.

The deafness was noted before the clinical appearance of the mumps, but this does not rule out mumps as an etiologic factor, for otologists occasionally find the involvement of the ear preceding the clinical manifestation of parotitis. However, while mumps can attack both ears, it does so to different degrees, and complete bilateral deafness is rarely seen after mumps.

The third possibility was the encephalitis. While Moe can find no reported cases of deafness following encephalitis, he holds this etiologic explanation to be the most plausible.

GROVE, Milwaukee.

Larynx

ROENTGEN THERAPY IN CANCER OF THE LARYNX. MAURICE LENZ, J. A. M. A. **134**:117 (May 10) 1947.

Good results depend on the administration of sufficient roentgen rays to destroy the cancer while preserving the recovery power of the simultaneously irradiated uninvolved larynx and laryngopharynx.

Tolerance of radiation injury diminishes with increase of the irradiated area. Small cancers are therefore more easily treated than extensive ones. Roentgen irradiation of small fields is readily tolerated by ambulatory patients. An ample zone of grossly uninvolved tissue surrounding the cancer, however, should also be adequately irradiated in order not to exclude silent extensions, which later may give rise to local recurrence and metastasis.

The fact that there are few lymphatic channels at the edge of the vocal cord as contrasted with the rest of the larynx, which presents a rich lymph capillary plexus, explains why cordal cancer is slower to extend and metastasize than growths of other portions of the larynx. Spread occurs at first by contiguity and along the delicate, horizontally placed lymph channels the length of the cord, more commonly forward and across the anterior commissure.

One is therefore confronted with the fact that as far as curability is concerned roentgen therapy of early cordal cancer in which the entire larynx is irradiated compares favorably with the more limited approach by laryngofissure and cordectomy.

Though many disagree in regard to this view, roentgen therapy may even be superior to this operation, especially if the cancer has extended beyond the cord and the anterior commissure. Roentgen therapy results in a much better voice than laryngofissure, a fact which will be admitted by any impartial observer who has had the opportunity to watch the results of both methods over a period of years.

Thus roentgen therapy is recommended in all cases of cancer of the vocal cord except those in which the cancer has extended subglottically or into the arytenoid cartilage. In the latter event laryngectomy followed by roentgen therapy is the best procedure, especially if the microscopic study of the growth indicates that the cancer was incompletely removed.

GORDON, Philadelphia.

SUPERVOLTAGE ROENTGEN THERAPY OF CARCINOMA OF THE ESOPHAGUS. FRANZ BUSCHKE and SIMEON T. CANTRIL, J. A. M. A. **134**:127 (May 10) 1947.

The authors present this discussion in an attempt to overcome the defeatist attitude that seems to prevail in the medical profession.

They have seen only 19 patients with esophageal carcinoma since 1939.

From the reports in the literature and from their own experience they feel that with 200 kilovolt radiation therapy a temporary reopening of the obstruction lasting from six to eighteen months can be expected in about half of the patients with carcinoma of the esophagus who are still in a sufficiently good general condition to support the procedure.

The histologic analysis of specimens of esophageal carcinoma substantiates the clinical experience that at least a proportion of them represent essentially radiovulnerable carcinoma similar to the radiocurable carcinoma of the larynx, the pharynx, the cervix or the oral cavity.

They discuss the value of irradiation of small fields in the million volt range, though they have to date only meager results to report.

GORDON, Philadelphia.

CONTACT ULCER OF THE LARYNX. GEORGIANA PEACHER, J. Speech Disorders **12**: 67 (March) 1947.

Contact ulcer of the larynx is a disease recognized as a clinical entity in 1928. It is characterized by typical signs and symptoms, has a definite location, cause and pathologic process and is of indeterminable duration. The lesion is most frequently overlooked altogether. When seen, invariably it has been mistaken for other laryngeal entities. Vocal abuse appears to be the exciting cause of the ulcer; the lesion is perpetuated by necrosis of the tip of the vocal process of the arytenoid cartilage located in the bed of the ulcer. Diagnosis is easily made by mirror and direct laryngoscopy. Biopsy may be necessary to exclude other laryngeal involvements in the final diagnosis. Present treatment indicates that cure is not possible without a long period of complete silence. Surgical treatment often facilitates the reparative process.

PALMER, Wichita, Kan.

Nose

CICATRICIAL STENOSIS OF THE NASOPHARYNX: CORRECTION BY MEANS OF A SKIN GRAFT. F. A. FIGI, Plastic & Reconstruct. Surg. **2**:97 (March) 1947.

Figi has found that cicatricial stenosis of the nasopharynx occurs with the same frequency in both sexes, that it is not confined to children and that excessive trauma in tonsillectomy, and not syphilis, is the most frequent cause. Other causes in his series were inflammation of unknown origin, rhinoscleroma, caustic burn, diphtheria and lupus. There was a variation in the duration of the stenosis of from two months to forty-four years. The degree of the stenosis varied from slight contracture to complete closure. The symptoms are mainly those of nasal obstruction. The patients are most often referred as having disorders of the accessory sinuses or the ears. Correction is achieved by freeing the scar attachments and dilation. Good results have been reported from simply making an incision through the occluding membrane, with no dilation. The originality of the author's method lies in the relining of the nasopharynx with a skin graft. A detailed description of his method is given.

SELTZER, Philadelphia.

INSTITUTION OF THE CHISEL FOR THE SAW IN RECONSTRUCTIVE SURGERY OF THE NOSE. W. L. GATEWOOD, Plastic & Reconstruct. Surg. **2**:149 (March) 1947.

Gatewood follows the usual methods in rhinoplasty with the exception that he discards the saw in favor of the chisel when cutting bone and sometimes cartilage. He finds that this saves time and energy, leaves smoother and cleaner bone surfaces, causes less injury of soft tissues and reduces the danger of osteogenesis, since there is no bone dust.

SELTZER, Philadelphia.

Miscellaneous

A PRELIMINARY INVESTIGATION IN THE TESTING OF LIP READING ACHIEVEMENT. GLADYS REID, J. Speech Disorders **12**:77 (March) 1947.

Sixty-two girls from the Lexington School for the Deaf in New York and 37 girls from the State School for the Deaf at Delavan, Wis., were selected for a lip-reading test on motion picture film. The children from the Lexington School were tested in groups of about 20. Each test consisted of (1) a series of 17 vowels and diphthongs, (2) a series of 11 consonants, (3) a series of 10 unrelated sen-

tences, (4) a series of related sentences telling a story, (5) a short story at the end of which were four questions which the subjects were to answer. At the school for the deaf in Wisconsin, the test was given to the entire group at the same time. The results of this study indicate low correlations, approaching zero, between lip-reading ability on the test used and other factors. From these results it is reasonable to conclude that lip-reading ability is not quantitatively correlated with, and cannot be predicted from, the length of training in lip reading, the mental age, the intelligence quotient or the grade status. However, it must also be considered that in this study only one factor in the total lip-reading situation, namely, articulation, was isolated and tested.

PALMER, Wichita, Kan.

A NEW METHOD OF HEMOSTASIS. VIRGINIA KNEELAND FRANTZ, New York Med. **3**:15 (April 5) 1947.

Dr. Frantz reviews briefly the history of hemostasis and cites the use of gel. film, gauze and foam from salts of alginic acid, beef thrombin and plain cotton. The latter has been used successfully in some 1,000 cases at the Neurological Institute in New York. The purpose is, of course, to create a clot in the wound by the use of some agent which will not be absorbed into the circulation, there to work harm to the patient. At this time there are three absorbable nontoxic and nonirritating hemostatics which may be applied directly to bleeding or torn vessels. They are: fibrin foam, gelatin sponge and oxidized cellulose—the last in the physical form of gauze or cotton. The "new method" of absorbable packing is now well established. This packing is sterilizable, adapts itself to the conditions present and can be saturated with the extract of brown seaweed—alginic acid—produced by hydrolysis. In places which are not amenable to suture—e. g., the liver and the pulmonary lobes, the retroperitoneal and retropleural areas—the use of oxidized gauze is safe and does not block drainage. It does not "stick" to the tissues, becomes gelatinous and fluid and can be removed as early as twenty-four hours after operation with impunity. "There is no evidence of any increase in infection."

The point is stressed that there is no substitute for careful surgical treatment, clamp and suture-ligature. However, no mention is made of the time-saving use of the electrocautery applied to the artery clamp instead of suture, and thus "cooking" the tiny area and sealing it.

VOORHEES, New York.

PLASMA FIXATION OF SKIN GRAFTS. J. E. SHEEHAN, Plastic & Reconstructive Surg. **1**:317 (Nov.) 1946.

Sheehan uses the dermatome for taking the skin grafts. He describes his method and gives the details of the preparation of the patient's plasma and of the securing of leukocytes for the white cell extract. When the donor area has been outlined and cleansed, the surrounding cutaneous surface is powdered with parlodian and sulfadiazine. The graft is excised, and the denuded area, after thorough cleansing and drying, is treated with an antibiotic. The adjacent surface of the skin is coated with rubber cement. The plasma preparation is applied to the area to be grafted, and the under surfaces of the graft are painted with the white cell extract. Sutures are not used, and only a linear scar results. With strict asepsis, the result is generally good.

SELTZER, Philadelphia.

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

Frank R. Spencer, M.D., *President*

H. M. Taylor, M.D., *Vice President, Presiding*

Sixty-Eighth Annual Meeting, April 23 and 24, 1947

Abstracted by Samuel Salinger, M.D.

The Pathology of Nasal Polyps. Presented by Dr. J. J. Shea, Memphis, Tenn.

Not all nasal polyps are necessarily of allergic origin. There are in the nose many varieties of polypoid growths of benign character which, by reason of location and, at times, a tendency to become cancerous, may have serious implications. Masses growing from the superior meatus may erode into the cranial cavity, with disastrous consequences. It is important, therefore, to section growths removed from the nose, with a view to determining their exact pathologic nature. A number of various types are presented, with sections showing their histologic characters.

DISCUSSION

Dr. Harry P. Schenck, Philadelphia: Billroth in 1855 made a classification of nasal polyps which has not been greatly improved on. His recognition of mucous polyps as a distinct entity was later supported by Zuckerkandl and Wacht. The latter also showed that all polyps, including carcinomatous, fibrous and adenomatous varieties, had a common origin in the mucous membrane and possessed all the components of the membrane and in abnormal relationship. Mucous polyps are the most common type found in the nose. Although their relationship to allergy is difficult to prove, the evidence is convincing. Zuckerkandl pointed out eight sites of origin of polyps, which agree with the ridges described by Van Alyea and which are frequently overlooked. They are in relationship to the turbinal structure, which is the shock organ of the nose responding to allergic irritation. Adjacent ethmoid cells become involved in the polypoid degeneration as the result of the spread of the vascular reaction originating in these tissues. It is important that all polyps or polypoid tissue removed from the nose be subjected to histologic study for the determination of the etiologic factors and the pathogenesis.

Dr. J. M. Robison, Houston, Texas: Polypus formation is a part of, or is related to, the attempt of nature to create and maintain a constantly changing environment of the body cells, that is, by the movement of the extracellular fluid, by active massage and gravity and, during the pathologic states, by the increase of tissue pressure and the transmission of the pulsations of the arteries to the tissue spaces and lymph channels to move the lymph or the extravascular fluid. In bony and closed cavities there are only the tissue pressure and pulsations to accomplish this purpose. This is explained more fully in my recent article in the Archives (**45**:405 [April] 1947). In the allergic state fluid is diffused into the tissues and frequently is not absorbed because of mechanical difficulties. This fluid is subject to infection, with subsequent fibrosis, resulting in polyp formation.

Dr. Burt R. Shurly, Detroit: It is important to study the individual patient in every case of polyposis. One is prone to exaggerate and attribute all polyps

to allergy. These tumors should be removed in order to afford the patient improved breathing space and to study the underlying pathologic changes. The patient is entitled to relief during the tedious process of establishing the role of allergy and administering proper measures for a cure.

Dr. Robert F. Ridpath, Philadelphia: Compared with the great number of people suffering from allergy, few have polyps. Other etiologic factors must be investigated. Experiments of a biochemical nature have shown that in many of these cases there is a deficiency of calcium as well as persistent leukocytosis. This suggests a deeper pathogenesis, necessitating much further study.

Dr. Samuel Salinger, Chicago: Much confusion arises from the fact that physicians have included under the term "nasal polyp" a variety of growths having different pathogenesis and pathologic characteristics. The fault is rather in the nomenclature and in the loose application of the word. The average physician considers a nasal polyp to be a pedunculated mass or group of masses which, he believes, are due to an allergic background. Tumors removed from the nose should be designated as cancerous or noncancerous until pathologic examination reveals their true characteristics, by which they may be recognized and properly named.

Dr. John J. Shea, Memphis, Tenn.: Bacteriologic studies have been carried out during the past year in the hope of uncovering something significant. The data thus far have proved inconsistent. It is hoped that further study along this line may be more productive.

Unusual Sinuses of Interest to the Surgeon. Presented by Dr. F. W. Dixon, Cleveland.

A colored movie is shown depicting the sinuses in various sections of the skull. Attention is directed to the wide variations in development and extension of the frontal, ethmoid and sphenoid sinuses. Encroachment of these cavities on the terrain of the others is so common that the term "normal" can seldom be employed except possibly in the case of the antrums, which are more frequently symmetric than the others. Careful study of the conformation and the extent of the sinuses is extremely important not only in the analysis of symptoms but in surgical procedures, in connection with which this information is paramount.

DISCUSSION

Dr. B. N. Colver, Los Angeles: Two cases of unusual sinus conformation are germane to the subject. In the first, the patient, who had asthma, was relieved by removal of a single polyp and splitting of a large turbinal structure, the tissues of which were cystic and contained in the lateral half two baby polyps which had not yet developed. There were no other cells or polypi. In the second case a draining fistula in the upper eyelid, due to an abscess following an injury incurred two years previously, appeared to be due to infection of a frontal sinus, as shown in the roentgenograms. Operation disclosed a normal frontal sinus with an infected ethmoid cell lying laterally which was the seat of the original infection.

Dr. Oliver E. Van Alyea, Chicago: Dr. J. Parson Schaeffer some years ago made the significant statement that variability of sinus conformation was so common as to be considered normal. Two adjoining sinuses, such as the frontal sinuses or the sphenoid sinuses, growing up together are extremely likely to override in one direction or the other, with consequent invasion, one of the other. In addition to osteoma appearing in the vicinity of the ostium of the frontal sinus and causing obstruction to drainage, one must not lose sight of the accessory cells

which trail along with the frontal sinus in its upward growth and which may cause distortion and block drainage.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: Among the abnormalities which I have seen were an infraorbital nerve entirely surrounded by bone, having no connection with the orbital floor, and an optic nerve entirely within the lumen of the frontal sinus, having no connection with the periphery except anteriorly and posteriorly. These and Dr. Dixon's cases show why conservative treatment fails in many cases because of abnormal conformations interfering with drainage, conditions that are disclosed only at operation.

Tumors of the Nasopharynx. Presented by DR. H. J. WILLIAMS, Philadelphia.

For accurate diagnosis of a tumor of the nasopharynx it is important to get a clear view with the postnasal mirror, as well as to employ the nasopharyngoscope. Nasopharyngeal cancer is most common between the ages of 40 and 60 and comprises 3 per cent of all malignant growths of the upper respiratory tract. Squamous cell carcinoma and reticulum cell sarcoma are the most common types encountered. Anaplastic carcinoma is usually associated with early involvement of the cervical lymph nodes, and frequently this constitutes the earliest indication of the process. Early involvement of the orifice of the eustachian tube gives rise to symptoms of auditory obstruction. Increased growth may lead to involvement of the orbit, the paranasal sinuses and the cranial cavity, with symptoms that are unmistakable. Many nasopharyngeal growths are radiosensitive and will regress considerably under appropriate radiation therapy. Three cases are presented.

(NO DISCUSSION)

(In closing, Dr. Williams stated that he had emphasized the importance of suspecting carcinoma when a patient has a swollen lymph node the enlargement of which cannot be clearly explained.)

The Surgical Treatment of Nasopharyngeal Fibroma. Presented by DR. J. M. BROWN, Los Angeles.

This is primarily a tumor of adolescence which tends to regress with maturity. It usually arises from the periosteum of the base of the sphenoid bone or of the basilar process of the occipital bone and may extend in any direction. It consists of a dense plexus of fibroblasts with numerous vessels. These account for the marked tendency to bleed. Six cases are reported in which fibroma was successfully removed through a transpalatine approach, with intratracheal administration of the anesthetic. Preliminary ligation of one or both external carotid arteries was frequently necessary for the control of hemorrhage. In some cases a loop was placed around the vessel but not tied unless indicated. The growth was separated from the base of the skull and removed in toto. Nasal extensions were found in all the cases. In only 1 case was there a residue. The palate was closed by a double line of sutures.

DISCUSSION

DR. PERRY G. GOLDSMITH, Toronto, Canada: The great danger in these cases is hemorrhage. In a case of this type I observed the patient die on the table as a result of the bleeding, which was not appreciated at the time because the suction apparatus was draining the blood into a bottle beneath the table. Mistakes are made and should be told as an antidote to undue enthusiasm over brilliant results. In another case of severe hemorrhage the wrong carotid artery was tied. The opposite artery was then ligated, and still the patient bled, leaving the surgeon in

a deep quandary. Fortunately, under high voltage roentgen rays the remnant of the growth disappeared, and the patient recovered. Radiation has a definite place in the therapy of these tumors and frequentlly accomplishes what surgery alone may fail to do. Nasopharyngeal fibromas are all malignant, whether termed fibroma or sarcoma. The nomenclature depends on the degree of the cellular component in relationship to the fibrous component of the mass.

DR. F. T. HILL, Waterville, Maine: The term "fibroma of the nasopharynx" may be a misnomer because, although the tumor grows from the fibrous tissue of the periosteum of the sphenoid bone, it is an extremely vascular mass. Perhaps the term "vascular fibroma" or "angiofibroma" would be more accurate. Extensive growth of the tumor may preclude the possibility of surgical extirpation, as demonstrated in a case in which it invaded not only the nasopharynx but the ethmoid labyrinth, the orbit and the floor of the anterior fossa, causing a spontaneous pneumatocele. Implantation of radon plus surgical diathermy produced an excellent result.

DR. JOHN H. FOSTER, Houston, Texas: Experience of years has demonstrated the advantages of radon implantation in cases of nasopharyngeal fibroma, so that surgical intervention is rarely found necessary. Certainly, the regression takes place without danger of hemorrhage, and when surgical excision becomes necessary to remove the residual mass, the influence of the irradiation is manifest in the slight bleeding that attends this delayed procedure. Several applications of radium made over a period of time may be required to accomplish these results, but the freedom from danger of hemorrhage makes it worth while.

DR. FREDERICK A. FIGI, Rochester, Minn.: Surgical removal of a juvenile basal fibroma is an extremely formidable procedure. The excellent results obtained by Dr. Brown in his series are noteworthy. In many cases the growth is so extensive as to be beyond the reach of surgical intervention. While radiation therapy yields satisfactory results to a certain degree, one must remember that the therapy is often followed by unpleasant sequelae, such as atrophy of mucosa with crusting. At the Mayo Clinic results have been more satisfactory when we used surgical diathermy in conjunction with radiation. A recent case exemplifies some of the difficulties. A young man of 18 had a tumor which filled the nasopharynx, the right nasal fossa, the antrum, the ethmoid cells and the retromaxillary fossa. The right cheek bulged markedly. Consequently, the tumor was approached through the antrum and destroyed as far as possible with surgical diathermy after ligation of the external carotid artery. Radon seeds were then implanted in the residual mass, and, despite the fact that Stenson's duct was draining into the antrum operated on, which gave rise to a suspicion that the discharge was cerebrospinal fluid, the entire condition cleared up after the closing of the defect of the anterior wall which had communicated with the mouth.

DR. J. M. BROWN, Los Angeles: I have no quarrel with those who wish to use radium. That therapy takes considerable time and, as has been stated frequently, is followed by atrophic changes. It probably is safer in the very young patient, in whom the tumor is more vascular. However, in older patients, in whom the tumor tends to regress, the surgical approach described has a definite place in practice.

A Study of the Eustachian Tubes: Their Orifices and Lumens. Presented by W. L. SIMPSON, Memphis, Tenn.

Five cases are described in which a large facial defect following exenteration of a tumor permitted one to view the upper surface of the soft palate, the movements of which were studied with respect to the effect on the orifice of the eustachian tube.

Contraction of the muscles of the palate, though hermetically closing off the nasopharynx, failed to affect the lumen of the ostium. Apparently, the tube remains open under all conditions.

DISCUSSION

DR. FRANCIS L. LEDERER, Chicago: To evaluate function properly, it should be observed under normal physiologic conditions. This obtains under the conditions represented in the cases shown. Is it not possible that the absence of change in the permeability of the eustachian tube during the muscular contractions observed may have been due to the absence of the normal hermetically sealed cavity?

DR. B. N. COLVER, Los Angeles: With altered anatomy one may reasonably expect altered physiology. In this instance it is my feeling that the opening or the closure of the tube takes place at the isthmus rather than at the orifice. Whether the opening or the closing is strictly the result of muscular action, or whether it is due to the pneumatic plus or minus change, is another question which should be considered.

DR. W. L. SIMPSON, Memphis, Tenn.: Answering Dr. Lederer, I would say that these examinations were made soon after operation and were confirmed repeatedly over a period of years. In the 5 cases cited, with hearing normal, the tube was not found closed at any time and its patency was not affected by swallowing or what not. Answering Dr. Colver, my observations show that the tube closes from the orifice to the isthmus, and its lumen is not affected by muscular action.

The Pathology and Surgery of Extrinsic Cancer of the Larynx. Presented by H. B. ORTON, Newark, N. J.

In 51 cases of extrinsic carcinoma of the larynx in which surgical excision was resorted to, this treatment resulted in 27 per cent five year cures. The growths involved the epiglottis, the aryepiglottic folds, the preepiglottic space, the piriform fossa and the postcricoid area, and their treatment required extensive resection of pharyngeal tissues as well as bilateral removal of the regional glands. Compared with the results of irradiation the surgical results are most favorable. Reported successes from irradiation of growths of this type, so frequently labeled "regressions," cannot be considered as cures when the five year limit is applied. Radium and roentgen rays should be used only when the patient refuses surgical excisions or when the lesion is clearly inoperable. Many patients who were given radiation therapy might have been saved had adequate surgical removal been carried out in the first instance. The pharyngostoma resulting from this operation can be closed by a plastic procedure at the appropriate time.

DISCUSSION

DR. FRANCIS E. LE JEUNE, New Orleans: This presentation should revise one's thinking in the matter of extrinsic carcinoma of the larynx. The results Dr. Orton has shown are amazing. The procedure is a heroic one, since most cases require complete block dissection of the neck. Jacobson stated long ago that in this type of case the life expectancy without surgical intervention was from twelve to fifteen months. Operation, therefore, even though successful in a minority of the cases, is certainly to be commended.

DR. HARRIS P. MOSHER, Marblehead, Mass.: I agree with the previous speaker that more men of this society should be operating in such cases. I should like to have Dr. Schall speak for me concerning the technic.

DR. LEROY A. SCHALL, Boston: Radical dissection of the glands in such cases has revealed the fact that many of them are the seat of metastases despite absence

of palpability. My associates and I have made it a practice to remove the sternohyoid muscles with the body of the hyoid bone in order to be sure that the lymph gland in the cricoid membrane is eliminated together with the rich supply of lymphatic channels in the muscle bed. Like Dr. Orton, we, too, are in favor of exploring the carotid sheaths. We feel that every intrinsic carcinoma of the larynx is potentially an extrinsic one. The greatest difficulty in all cases is the selection of a proper procedure. Radiation when properly applied by an expert has a definite place in our therapy. Our records reveal cures in 24 per cent of patients with extrinsic carcinoma treated between 1930 and 1940. It certainly should be employed when the patient refuses radical surgical excision or when his general condition contraindicates the latter. Finally, it is very important that all patients be followed up with yearly examinations for at least ten years.

Dr. Louis H. Clerf, Philadelphia: There are unfortunately too many radiologists who think in terms of palliation, reporting results after two or three years. My colleagues and I believe that the five year limit should be considered minimal. Furthermore, where irradiation has failed it has been our experience that subsequent surgical treatment is more difficult and the results not as good as if there had been no preceding radiation therapy. There is still some confusion in regard to the terms "intrinsic" and "extrinsic." The decision between thyrotomy and laryngectomy depends not so much on whether the lesion is restricted to one cord as on the degree of infiltration and fixation, indicating extension to the cartilage itself. As for the truly extrinsic growth involving the epiglottis, its folds, the arytenoid cartilages, one must be radical through sufficiently adequate exposure. The object is to save the patient's life, disregarding for the moment the tissues which must be sacrificed.

Dr. Frederick A. Figi, Rochester, Minn.: I, too, favor radical surgical intervention in the type of case described by Dr. Orton. While it is true I have seen several extensive cancers apparently retarded for three to five years, I still feel that better results can be obtained through procedures such as those described by Dr. Orton.

Dr. Edwin N. Broyles, Baltimore: There was a time when if one found, while operating on a larynx, that the carcinoma had extended through the cricothyroid membrane, one would back out and turn the patient over to the radiologist. Today one follows the lead of the general surgeon and proceeds to remove all the diseased tissue, including glands and muscles. (Several microscopic slides were shown revealing that the carcinoma had extended into the ligament of the cord and into the cricothyroid membrane.)

Dr. Charles J. Imperatori, New York: I agree with Dr. Orton and the others regarding the necessity of radical surgical excision in the type of case previously considered inoperable. However, I believe laryngologists should give more thought to the problems of cancer research, since there is hope that through metabolic, biochemical or endocrine mechanisms as yet undiscovered, the way may be paved for a therapy that will in the future make surgical excision unnecessary. Until that day arrives one must employ every means at hand, including the radical surgical operation and irradiation, thoroughly, courageously and skilfully, as demonstrated by Dr. Orton.

Dr. Henry B. Orton, Newark, N. J.: Answering Dr. Imperatori, I do state, in a portion of the paper which, for lack of time, I was unable to read, that the objective is complete destruction of the cancer, whether by surgical operation, irradiation or some as yet undiscovered biologic or biochemical process. Concerning the definition of "extrinsic," my associates and I consider any lesion above

the vocal cord as in that category. If the growth is on the epiglottis, its folds, the arytenoid cartilages, the piriform sinus or the pharyngeal wall, we term it "epilaryngeal." It must be recalled also that a lesion beginning as extrinsic may also become intrinsic, as well as vice versa.

I showed slides representing 48 patients, 18 of whom are alive and well five years or more after operation. That is a little over 40 per cent.

Hemangiomas of the Mouth. Presented by DR. FREDERICK A. FIGI, Rochester, Minn.

The types of hemangiomas encountered about the oral cavity are the capillary, the cavernous and the racemose. The capillary type is not seen so frequently as the others except in cases in which a "port wine" nevus extends from the face into the lip or the adjacent oral mucosa. Small capillary growths may respond to radium pack, but genuine "port wine" neoplasms when so treated often undergo an active dermatitis and may become malignant. They require wide excision and extensive repair. Women, by the use of cosmetics, may successfully avoid the necessity of surgical treatment.

The cavernous type may occur anywhere in the oral cavity and is often troublesome because it bleeds easily when traumatized. Macrocheilia and macroglossia are particularly dangerous. They are best treated by implantation of radium seeds in the case of infants and children and by injection of boiling water, the use of sclerosing agents or electrocoagulation in the case of adults. Wherever possible, the electrode is introduced through the intact skin. It must be well insulated, since an intense heat is required. This procedure is particularly useful if there is a large hemangioma of the tongue or the lip. It is important to avoid the branches of the facial nerve.

Phleboliths are occasionally encountered, which give rise to considerable pain, especially if located over bony prominences of the face.

Racemose or plexiform angiomas, characterized by expansile pulsation, increased on straining, require ligation of the afferent vessels.

(NO DISCUSSION)

The Thyroid Gland and The Nose. Presented by DR. A. W. PROETZ, St. Louis.

Eighty-four patients who had chronic nasal disease of the allergic type were studied from the point of view of thyroid deficiency. Thirty-four of the patients were known to have had hypothyroidism. The dominant characteristics of all of these patients were indolence, unresponsiveness, asthenia and dry skin. The nasal mucosa was not always of the pale, boggy type, there being many instances in which it was red and turgescent. Of 50 cases in which hypothyroidism was unsuspected, the basal metabolic rate was —10 to —20 per cent in 18, —30 per cent in 8 and —40 per cent in 4. The results of thyroid therapy were encouraging; 37 patients definitely improved, 35 showed doubtful results and 12 showed no improvement. The basal metabolic rate was not always reliable, and in some instances it was ignored. Therapy was administered on the basis of the history and the findings, and the encouraging results seem to point to a nutritional factor in the etiology.

(NO DISCUSSION)

The Present Status of Diphtheria. Presented by DR. E. H. PLACE, Boston.

Although the number of cases of diphtheria has diminished in Boston from a high of 150 to 30 per hundred thousand, the percentage of complications, cardiac

and muscular, as well as the mortality rate, remains practically unchanged since the advent of serum. The mortality rate depends greatly on the virulence of the organism, since it has been shown that the gravis type has yielded as high a rate as 15 per cent, whereas the mitis type has yielded as low a rate as 0.5 per cent. The toxin seems to be the same in all types. Higher mortality also is the result of delay in administering antitoxin in view of the insidious nature of the disease in some epidemics.

Immunization is only relative. There is no way of ascertaining in advance the degree of immunity that may be expected from toxoid or toxoid-antitoxin. As a rule, the toxoid is better for children, whereas the toxoid-antitoxin works best in adults. Booster doses at three to five year intervals have proved helpful.

Antibiotics are of value in the treatment of carriers; 200,000 units of penicillin given intramuscularly every day for one week will clear up 95 per cent of the throats of carriers.

As for the cases of laryngeal diphtheria, good results have followed direct intubation, according to the Mosher method, as practiced in Boston. Intubation fatalities are in most cases due to secondary pneumonia, which can be prevented by the use of antibiotics. Not enough cases have been seen to warrant conclusions as to the relative merits of intubation and tracheotomy.

DISCUSSION

DR. HARRIS P. MOSHER, Marblehead, Mass.: You have heard from one of Boston's best physicians, and to my many obligations to him of the past may be added this one of today, namely, his coming here to give members of this association the benefit of his ripe experience.

DR. BURT N. SHURLY, Detroit: My interest in this topic is more than academic, since I happen to be the survivor of three children afflicted with this disease. I have seen all the marvelous progress in the therapy of diphtheria from the preantitoxin days to the present, and the memory of the numerous intubations, with all the trials and tribulations involved in the care of these patients, is still vivid; likewise, the improvement in results following earlier diagnosis and the employment of larger initial doses of antitoxin. Of greatest importance has been the widespread resort to immunization of children. Laryngologists must never relax their vigilance in this direction. I believe that intubation is still a proper procedure. A tracheotomy can always be done, should the former prove inadequate.

DR. GORDON F. HARKNESS, Davenport, Iowa: I should like to have Dr. Place discuss those membranous inflammations in which the organisms are identified as Corynebacterium pseudodiphtheriae, Hoffmann's bacillus or Bacillus erodiens. It is my understanding that as nonvirulent organisms they are not variants of the virulent organism at any time. From my meager knowledge it appears that the only method of positive identification is that of inoculation of guinea pigs. The possibility of becoming legally entangled through mistaken quarantine in a case of a nonvirulent membranous lesion must be considered. I know of such a case.

DR. PAUL H. HOLINGER, Chicago: The incidence of diphtheria in the Children's Memorial Hospital of Chicago is so low that physicians frequently forget it. It certainly must always be kept in mind with children admitted because of laryngeal obstruction. I should like to know what Dr. Place's experience has been relative to the present age incidence. I have seen a 22 month old infant admitted in a moribund condition due to overwhelming diphtheria; also, death from the same cause in a 2 month old infant.

DR. HENRY B. ORTON, Newark, N. J.: The question of chronic stenosis following intubation is important. Years ago I fell heir to quite a number of cases of this type. The questions I should like to ask Dr. Place are: "How often do they intubate at the Boston City Hospital? If the tube is coughed up once or twice, will they reintubate in preference to doing a tracheotomy?" My colleagues and I have found that we encounter no stenosis since we established the rule that if the patient expels the tube two or three times we will do a tracheotomy.

DR. FREDERICK T. HILL, Waterville, Maine: I should like to ask Dr. Place whether or not the program of mass immunization, perhaps inadequately carried out, might tend to decrease the natural immunity of the individual person.

DR. H. MARSHALL TAYLOR, Jacksonville, Fla.: The fact that one does not see diphtheria as often as previously is all the more reason why one should keep his eyes open. Only recently I saw a child suffering from epistaxis who had paralysis of the lower extremities. Yet several physicians had missed the diagnosis. I should like to ask Dr. Place whether he has seen a higher incidence of paralysis in the cases of nasal than in the cases of pharyngeal diphtheria and whether he has any suggestions as to therapy in this complication. I have seen 3 children with paralysis of the lower extremities who seemed to get along very well on a velocipede, as did the child first mentioned.

DR. E. H. PLACE, Boston: The coughing up of the intubation tube has always been a problem. However, at Boston City Hospital we have found fewer instances of this complication since using the Bayer tube in place of the O'Dwyer. Naturally, much depends on selection of a tube of the proper size, as well as on the skill of the physician.

As for toxoid versus toxin-antitoxin, we have found the former preferable for children and the latter for adults. Concerning membranous infections not truly diphtheria, we feel that there is no basis for assuming that there is a nonvirulent diphtheroid infection. Diphtheroids produce no disease. They are prevalent everywhere and have no pathologic significance. As has been stated by some of the speakers, one cannot emphasize too strongly the importance of giving antitoxin early in adequate doses, even when the diagnosis is not definitely established. The possibility of diphtheria being present in association with other infections must also be kept constantly in mind. Dr. Holinger spoke of age incidence. We have not noticed any perceptible change in that respect. Certainly the age of the patient is no criterion of any value. It is possible, however, that the apparent increase of cases among adults may be due to general immunization in childhood. In the case of smallpox this was more apparent.

As for stenosis due to pressure of the tube as mentioned by Dr. Orton, we believe the factors are those previously mentioned, namely, too large a tube or unskilful manipulation. The case is different when the obstruction is due to streptococcic infection. Here the danger of perichondritis is great, and the patient is safer having a tracheotomy. In our own experience, however, the incidence of post-tubal stenosis in cases of diphtheria has been definitely much lower than formerly.

Concerning the paralyses mentioned by Dr. Taylor, we have never had residual paralysis in cases of diphtheria unless there was cerebral damage. Thrombosis may occur in the brain, and the resulting paralysis will be permanent.

We are strongly in favor of mass immunization on a universal plan as the only real safeguard. There is no evidence to show that immunization lowers the

immunity that develops under natural conditions. In fact, the case is entirely the opposite. The number of carriers has dropped markedly since immunization has been widespread.

The question whether the disease can be recognized by its odor depends on the keen sense of the observer. Diphtheria, as well as the other infectious diseases, has a marked odor, sometimes thought to be characteristic. However, when it comes to the real test, recognizing the odor when one is blindfolded, I have never been able to substantiate the claim. I believe such odors as do appear are due to secondary invaders, such as saprophytes. They certainly are not present in diphtheria cultures.

Tissue Changes Occurring in Sinus Membranes After Treatment with Penicillin. Presented by DR. R. A. FENTON, Portland, Ore.

The effects of penicillin on experimentally induced virulent sinusitis are reported. Six cats were infected by instilling a culture of Streptococcus pyogenes into the frontal sinuses, which were then treated with solutions of penicillin. Subsequently the animals were killed, and the tissues were removed for study. The solution employed averaged 2,000 units per cubic centimeter. It was found that in the treated animals the surface bacteria were rapidly destroyed and that fewer bacteria were present in the goblet cells and deeper tissues than in the control animals. However, it was noted that the acute inflammation persisted longer in the treated animals.

DISCUSSION

DR. A. W. PROETZ, St. Louis: This paper is important because it deals with a subject on which there has been so much unscientific generalized superficial observation. My findings have been the same as Dr. Fenton's so far as the clinical use of penicillin is concerned. I feel that in cases of chronic sinusitis it is practically useless. In the cases of acute sinusitis it probably has considerable value. I am not too sure that it is more effective than some of the older treatments; yet it certainly has a place in the treatment of acute sinusitis. One must remember that there is a difference between treating the nose and treating the sinus. The time element is important, because fluid remaining in the sinus over a period can do damage which fluid in the nose will not do, since the latter does not stay in the nose long enough. My own observations on the effects of penicillin have shown that as the concentration is increased beyond 250 units per cubic centimeter the duration of the ciliary beat is reduced. This is important, since ciliary activity corresponds to the viability and physiologic response of the cell itself.

(In response to a query from Dr. Imperatori, Dr. Fenton stated that he had had no experience in the use of the so-called aeropenicillin.)

(NO FURTHER DISCUSSION)

Chordoma of the Cervical Region. Presented by DR. C. J. IMPERATORI, New York.

Chordoma is a rare tumor usually found at the extremities of the notochord, the spheno-occipital and the sacrococcygeal. The diagnosis is made on biopsy, which reveals a syncytial giant cell and concentric nests of cells with nuclear vacuolation, the nucleus being eccentrically placed and the cell substance traversed by a fine trabecular network enclosing a glycogenic material. One case is reported. The

mass was found to the right of the midline, extending from the level of the epiglottis downward. Roentgenograms showed it in the region of the third to sixth cervical vertebrae. There was no bony involvement. It was removed by sharp dissection under suspension laryngoscopy. There was comparatively little bleeding, and the postoperative course was uneventful. The diagnosis was confirmed by three pathologists.

DISCUSSION

DR. J. J. SHEA, Memphis, Tenn.: Tumors of this type are rare, and the pathologic diagnosis is often a source of argument. In the case I wish to report, the specimen was studied for nearly three months before a satisfactory conclusion could be reached. The patient was a woman whose chief complaint was vertigo. The tumor was found in the region of the sphenoid sinus. Attempted biopsy via the postantral route resulted in trauma of the internal maxillary artery, necessitating ligation. This patient has been under observation for nineteen years, and regular roentgenologic studies have revealed no increase in the size of the tumor. It fills the sphenoid cavity and extends into the ethmoid cells. The type of cell found in the biopsy specimen had a very foamy cytoplasm and an intercellular material not truly cartilaginous.

DR. ROBERT F. RIDPATH, Philadelphia: At the time I reported 2 cases before this association I could find only 3 cases described in the literature. In the first case there was a history of a blow on the head causing unconsciousness. There were no symptoms for six months, when nasal obstruction and bleeding were observed. The bleeding continued to the time of the patient's death. Vertigo was a prominent symptom, as it was in Dr. Shea's case. I have seen 3 cases altogether, and in all of them ocular symptoms were present, leading to eventual blindness.

In such cases the history is important and emphasizes the need of careful examination of the epipharynx.

DR. EDWIN N. BROYLES, Baltimore: I should like to report a case. The patient was a 21 year old Peurto Rican girl. Her first intimation of the trouble was the appearance of a mass in the pharynx. It looked like a cyst, but when incised it was found to extend through the anterior vertebral ligament. Though not encapsulated, it came out easily and looked like fat. Section showed it to be mucous. (Slides were shown, illustrating the sections, as well as photographs of the pharynx.)

DR. C. J. IMPERATORI, New York: Dr. Broyles' case was similar to mine, except that the tumor was higher up, in a location where pressure symptoms did not appear. His slides showed typical celliferous cells, that is, large vacuolated cells with the nucleus at the periphery. In my case the mass was lower down, extending from the third to the sixth cervical vertebra. The thing I wish to stress is the confusion in diagnosis. It would seem imperative, therefore, that more than one pathologist examine the specimen. In my case it was seen by three men, and eventually the original diagnoses of mixed tumor and leiomyoma were reversed, when the true nature of the growth was revealed through further examination and consultation among the three men.

PRESENTATION OF INSTRUMENTS

DR. CHARLES J. IMPERATORI, of New York, showed an improved laryngeal loop attached to a regular head mirror, permitting the use of either eye and affording a magnification of 3.5 diopters. The apparatus can be rigged up by having a swivel attached to an ordinary optical loop. The head mirror is fitted into the swivel.

Dr. Edwin N. Broyles, of Baltimore, presented a bronchoscope having a small metal tube attached to its orifice through which a stream of compressed air may be passed across the opening of the tube. By this means the exhaled air is blown away from the operator, and this prevents fogging of the glasses.

Rhinoscleroma in Two Native-Born Brothers. Presented by Dr. D. S. Cunning, New York.

The details of 2 cases of rhinoscleroma are given. The first patient was observed over a period of five years before he succumbed to asphyxia when his tracheal tube became obstructed. The other has been under observation for over two years and also has to wear a tracheal cannula because of laryngeal obstruction. Histologic sections are presented, with a description of the various characteristic findings. The author has observed 8 cases of rhinoscleroma over a period of years but was never able to substantiate the claim of others that this condition is probably associated with tuberculosis.

DISCUSSION

Dr. I. H. Blaisdell, Syracuse, N. Y.: (The discusser presented a case of his own and expressed gratitude to Dr. O. D. Chapman, of Syracuse University, for his help in the bacteriologic studies.)

According to Dr. Chapman, who is an expert in this phase of bacteriology, Klebsiella type C is always found in rhinoscleroma. This germ has been called the Friedländer bacillus, the Frisch bacillus and Bacillus mucosus capsulatus by other observers. Despite this finding, one has to agree with Dr. Cunning that the true etiologic agent of rhinoscleroma is still uncertain.

Streptomycin was used in my case, and the "kodachrome" slides taken over a period of time show a definite diminution in the size of the lesion.

Dr. Fred W. Dixon, Cleveland: It is interesting to have reports of the progress of these cases over a period of years. I have had a case under observation for thirteen years, during which time I have had to remove granulation masses from the trachea six times, removal being followed with roentgen therapy; likewise, masses were removed from the nose several times. When seen recently the patient was feeling well and was able to breathe well. I believe that radiotherapy is helpful in making these patients comfortable, even though it cannot cure them.

Dr. LeRoy A. Schall, Boston: I have followed a patient for fifteen years, who has had irradiation of the rhinoscleroma and a plastic operation, and still the disease continues to progress. This disease is prevalent in the Latin-American countries, and it is likely that it may be seen more frequently here, owing to rapid transportation and increased intercountry travel.

Dr. Blaisdell's report of the favorable influence of streptomycin is indeed heartening, since up to the present time neither surgical procedure nor irradiation has proved to be more than palliative.

Dr. D. S. Cunning, New York: Dr. Blaisdell's pictures showing the favorable influence of streptomycin were extremely interesting.

In the first series which I reported five years ago, sulfonamide drugs were employed in 6 cases, and an initial improvement was noted. This, however, was due merely to control of secondary infection. The original lesions failed to show any change. In the last case, just reported, penicillin was used, without much success. Now that Dr. Blaisdell has had such success with streptomycin, I certainly intend to try it.

Pulsion Diverticulum of the Hypopharynx and Its Surgical Treatment. Presented by DR. C. L. JACKSON, Philadelphia.

Zenker, who first described this condition, was under the impression that it was due to a tear in the musculature of the hypopharynx. Today laryngologists know that it is in reality a herniation through a weak triangle formed by the converging longitudinal muscle fibers just above the cricopharyngeus muscle. This anatomic factor was recently well described by Brandenburg. In addition, it is believed that failure of the cricopharyngeus muscle to relax at the proper moment in deglutition acts as a predisposing factor. The symptoms and the diagnosis are well known. While several surgical procedures have been employed at various times for the eradication of the sac, my associates and I have had our best results from a one stage operation with or without the esophagoscope. Two hundred patients have been operated on by this method in our hospital, with only a 1 per cent mortality. The operation is done under block anesthesia induced by intravenous injection of some pentothal sodium. The incision is along the sternocleidomastoid muscle, care being taken to avoid the lobe of the thyroid gland. The esophagoscope is useful for checking the location of the sac and keeping it clean. There was only 1 case of recurrent paralysis.

(NO DISCUSSION)

Bronchoscopy in the Diagnosis and Treatment of Bronchiectasis in Children. Presented by DR. D. E. S. WISHART, Toronto, Canada.

Bronchiectasis can be cured in most cases by adequate surgical treatment. This must be based on thorough diagnosis, which includes adequate bronchography. Unless every segment of both lungs is properly outlined, one can never be sure of the extent of the disease. Mapping of the lingula, for instance, is often inadequate, and this is frequently the reason for failure after surgical intervention.

My procedure is to delineate one lung at a time. It is done under fluoroscopy, the patient having been anesthetized with ether, after preliminary sedation, through a small bronchoscope. Spot mapping without the Bucky diaphragm is followed out with taking of films as each segment is filled. The various positions for filling the different segments are described and illustrated.

This procedure has proved eminently satisfactory with nearly 400 children.

DISCUSSION

DR. LOUIS H. CLERF, Philadelphia: I have been converted to the need for general anesthesia in these cases after observing failures following surgical intervention, due to inadequate preliminary bronchography. If on the basis of the information given him preoperatively the surgeon fails to remove all of the diseased lung, one must conclude that something was wrong with the bronchography. We appreciate the need for giving meticulous attention to details and the importance of cooperating closely with the roentgenologist. I am not familiar with the intubation tube Dr. Wishart uses for the ether feed. Otherwise, our procedures are similar. We aspirate all secretions and pass a nasal catheter into the trachea, where it is guided under fluoroscopy to the desired portions of the tree.

DR. ROBERT L. GOODALE, Boston: Dr. Wishart's paper is timely because it emphasizes the importance of early diagnosis. In one third of all cases bronchiectasis arises in the first decade of life, and frequently adequate therapy is not applied until much too late.

I agree that the danger of general anesthesia has been exaggerated with regard to this procedure. Certainly, if the thoracic surgeon can operate with the patient

under general anesthesia, I can't see why it should not be as safe to have the patient under general anesthesia for the diagnostic procedure. I should like to ask Dr. Wishart whether he has used other methods of inducing anesthesia, such as intravenous or rectal injection.

Dr. Lyman G. Richards, Wellesley Hills, Mass.: I have had the pleasure of working with Dr. Wishart and appreciate the meticulous care with which he follows out the details which he described. I also agree that general anesthesia is safe and by far the preferable type, provided that the entire operation is carried out in one room. Moving the patient from the anesthesia room to the roentgenologic department involves a risk and diminishes the chance of success.

Dr. C. L. Jackson, Philadelphia: I am in complete agreement with Dr. Wishart as to the importance of bronchography, one lung at a time, so that every segment may be adequately outlined. Proper teamwork undoubtedly accounts for the success which he has attained. It is not always obtainable. As a rule, I do not favor general anesthesia, having been in the habit of doing this work with the patient under local anesthesia after some premedication. Use is made of the nasal catheter guided into the bronchial tree under fluoroscopy. Occasionally it is necessary to pass the catheter directly through the laryngoscope.

Dr. Paul H. Holinger, Chicago: Speaking of general anesthesia brings up the question of the building codes of various cities. In Chicago we are not permitted to use general anesthesia in any x-ray room where the apparatus is not only shockproof but explosion-proof as well. This setup is often difficult to obtain.

Dr. D. E. S. Wishart, Toronto, Canada: Replying to Dr. Goodale's query, I have had no experience with other methods of inducing anesthesia. I have followed the advice of the anesthetist and thus far have received no encouragement to deviate from the established methods. Dr. Holinger's point as to safety is well taken. In Toronto there has been no trouble in meeting the requirements of the building code.

The intubation tubes I use are little metal cylinders about 2 inches (5 cm.) long and open at both ends, introduced by means of a long straight steel tube, which is withdrawn after the small tube is properly placed. The pictures show them as being bent, an appearance which is difficult for me to explain.

I expected Dr. Jackson to discuss his method, which differs from that which I have described. Since my associates and I work in an antiquated building it is likely that our setup is different. Were we to achieve a more modern setup I should be happy to visit Dr. Jackson and see if I could not profit by his experience.

Book Reviews

Diseases of the Nose and Throat. By Charles J. Imperatori, M.D., and Herman J. Burman, M.D. Pp. 552. Price $12. Philadelphia: J. B. Lippincott Company, 1947.

This book is indeed a fine, concise and accurate textbook for senior students, interns and general practitioners. It lacks, however, the encyclopedic approach, so necessary for the serious study of otolaryngology.

The many illustrations are simple and admirable; the photographs are well taken. There is a certain sparseness of words, which is refreshing and leads to the compactness of the volume.

No man could expect to learn the minutiae of anatomy or physiology or surgical technic from its 552 pages. It is essentially a book for clinical evaluation and treatment and as such should be popular with the senior student. However, a post-graduate student must delve further into cause and effect and be prepared to discuss the theoretic concepts of otolaryngology and its relation to the rest of the specialties much more than this book does.

For rapid reference and good, solid therapy in the office and at home, it should hold high value and could be added profitably to any medical man's library.

News and Comment

MEETING OF AMERICAN OTORHINOLOGIC SOCIETY FOR ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

The American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery will hold its next meeting at the New York Academy of Medicine Building, New York, Thursday, Nov. 13, 1947, at 8 p. m. Programs may be obtained by addressing the Secretary.

A dinner will precede the meeting, at 6 p. m., at Hotel Plaza, Fifty-Ninth Street and Fifth Avenue, New York.

Reservations may be made with Dr. Norman N. Smith, secretary, 291 Whitney Avenue, New Haven, Conn.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Chicago. Time: June 21-25, 1948.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Millard F. Arbuckle, 539 N. Grand Blvd., St. Louis.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Old Homestead, Va. Time: April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 15 Whiting Rd., Wellesley Hills, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Chalfonte-Haddon Hall, Atlantic City. Time: April 7-9, 1948.

SECTIONS:

Eastern.—Chairman: Dr. J. Winston Fowlkes, 12 E. 63d St., New York 21.
Place: New York. Time: Jan. 16, 1948.
Southern.—Chairman: Dr. William A. Wagner, American Bank Bldg., New Orleans.
Place: New Orleans. Time: Jan. 23, 1948.
Middle.—Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Place: Columbus, Ohio. Time: Jan. 19, 1948.
Western.—Chairman: Dr. Meade Mohun, 23–2d Ave., San Mateo, Calif.
Place: San Francisco. Time: Jan. 31-Feb. 1, 1948.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Place: Old Homestead, Va. Time: April 12-13, 1948.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.